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WATER QUALITY CRITERIA DATA BOOK. VOLUME 4. AN INVESTIGATION INTO RECREATIONAL WATER QUALITY

B. J. Mechalas, et al

Envirogenics Company
El Monte, California

April 1972

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Water Quality Criteria Data Book
Volume 4

An Investigation into Recreational Water Quality

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27 Abstract

A new technique has been developed for establishing firm criteria for health risks associated with recreational water bodies. The basis of the method is a mathematical treatment of medical dose-response data in conjunction with the probability of exposure over a period of time to a given level of the potentially harmful factor, such that a quantitative risk can be assigned to the recreational activity. Once a public health jurisdiction has established an acceptable level of risk, curves produced by electronic data processing equipment can be used to ascertain whether a particular water should be open to the public. (Wilson-Envirogenics)

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Water Quality Criteria Data Book, Vol. 4

AN INVESTIGATION INTO RECREATIONAL WATER QUALITY

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ABSTRACT

The Envirogenics Co., under sponsorship of the EPA, has developed a new technique for establishing firm criteria for health risks associated with recreational water bodies. Initial analysis of data required in this methodology has demonstrated that scientifically valid standards for recreational water quality can be formulated that should replace the present rather arbitrary standards.

The basis of the method is a mathematical treatment of medical dose-response data in conjunction with the probability of exposure over a period of time to a given level of the potentially harmful "factor" such that a quantitative risk can be assigned to the recreational activity. Once a public health jurisdiction has established an acceptable level of risk (perhaps in association with Federal quality guidelines), curves produced by electronic data processing equipment can be used to ascertain whether a particular water should be open to the public.

While sufficient data have been found on both the health effects and the distribution of key factors to verify the effectiveness of the recommended procedure, informational gaps prevent the immediate adoption of the system. The gathering of information to establish realistic standards for key health-oriented factors would be an undertaking that could be accomplished in a relatively modest program. Once the essential information is obtained, it will be possible to put into practice the new Envirogenics-developed criteria procedure with the most critical factors. This advancement would be of great significance to the entire field of water quality standards.

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SECTION I

CONCLUSIONS

1. The factor to criteria conversion methodology developed permits the assessment of risk to recreationists entering waters with various hazard distributions.

2. The methodology of utilizing medical dose response data and factor concentration in the water as inputs into a mathematical model objectively develops risk curves.

3. The mathematical model developed for computer programming greatly facilities the handling of the collected data.

4. Water quality standards should be based on acceptable levels of risks to the user population. These can then be translated into specific factor concentration requirements for each water under an agency's jurisdiction.

5. Most State standards have no specific experimental foundation for their requirements. Most refer to other standards to the experience of others, or to one or two classical publications in the literature.

6. Contrary to several published views, Salmonella typhosa is not a significantly more infective pathogen than are the other Salmonella species.

7. The total coliform or fecal coliform concentration in a water must be exceptionally high ($\sim 8 \times 10^5$ MPN/100 ml water) before a significant level of risk (1 case/10^5) is faced by a recreationist.

8. In a given polluted water, the virus-coliform ratio, rather than the Salmonella-coliform ratio, will probably be the determining parameter in the setting of coliform standards.

9. In general, pesticides cannot be considered significant recreational water hazards. In most cases, and especially for the chlorinated hydrocarbons, the concentrations constituting risks are far in excess of water solubility.

10. Temperatures between 60°F and 93°F can be considered as the range for ordinary recreational usage. The recreationist's over-estimation of his own capabilities at water temperatures outside this range poses a real hazard.

SECTION II

RECOMMENDATIONS

The present study has been able to arrive at several specific conclusions based on the available data. The methodology can be considerably strengthened by deriving additional data that can serve either as basic inputs into the methodology, or to increase the level of significance of the available information.

Basic to this analysis of recreational water criteria is the assumption that the average recreationist imbibes 10 ml of water during his contact with the water. This figure is critical in deriving the level of exposure and should be confirmed experimentally.

Data quantifying the concentrations of Salmonella organisms in waters were difficult to find. Because the assumption that one Salmonella organism in water constitutes a health risk is not a valid one, more definitive studies of Salmonella distribution are needed.

Very little is known about the Salmonella and total coliform or fecal coliform ratios in polluted water. Studies to determine the relative distributions of these organisms in different types of waters are recommended.

The methodology should be refined to consider factor interactions. Specific recreational areas should be analyzed using the methodology and long term epidemological studies. Comparisons of actual and predicted risks can then be made, thus establishing a basis for quantifying factor interactions.

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SECTION III

INTRODUCTION AND SUMMARY

The assessment of the risk of ill effect faced by a population using various waters for recreational purposes has long been a problem for regulatory agencies. On the one hand, it was recognized that several serious diseases were associated with contaminated waters, and on the other hand, there was only the most tenuous of epidemiological evidence linking the use of such waters with disease. Agencies charged with safeguarding the public health have had to set standards for recreational waters, restricting their use when a dangerous level of a disease factor was considered to be exceeded. These standards were quite often based on inferences drawn from drinking water standards. The Envirogenics Company has undertaken an EPA-sponsored program to develop a methodology for establishing more rational criteria for health risks associated with recreational water quality (RWQ).

In order to meet RWQ standards, it may be necessary that pollution sources be controlled. In many cases, this will involve treatment of influents being discharged into recreational waters. Because the costs associated with treatment are often high, it is important that the RWQ standards be carefully defined so that treatment requirements do not become unachievable. The RWQ standards must also be based on criteria that permit regulatory agencies to estimate the level of user risk associated with recreational water bodies covered by such standards.

Water quality criteria should be based on objective data that relate concentration of organisms or chemicals to degree of risk. Unfortunately, this approach has not been followed in the past. Most RWQ standards are based on evidence of past fecal contamination and are derived from drinking water quality standards. Such RWQ standards have, however, withstood the test of time (60 years) and have contributed to the dramatic decline in water-borne bacterial diseases. Thus such standards cannot easily be abandoned by those responsible for safeguarding the public health.

The demonstration that coliform organisms were indicators of fecal contamination and the development of relatively simple techniques for their detection resulted in the development of microbiological water quality standards. However, as the more dramatic and explosive type of epidemics that had come to characterize water-borne infections decreased in occurrence, other problems began to increase. These new problems are due in part to (1) the development of huge population centers that overstress existing sanitation systems; (2) the introduction of an ever-widening spectrum of new chemicals that eventually end up in water; (3) the continual reuse of water as it travels down a stream course, resulting in a build-up of refractory materials; and, (4) the development of sophisticated instrumentation and epidemiological techniques that have identified previously unsuspected disease sources as being water-borne.

Epidemiologists have begun to question their previous thinking on what constitutes evidence of a water-borne incident. Many infections and toxicities are subclinical in nature, producing only relatively mild and brief illness. Such incidents usually go unreported, even though absenteeism from work and school results. There is also evidence that water-borne agents may cause a few direct disease cases among water users who in turn act as secondary sources of infection among the population at large. The distribution patterns of such transmitted outbreaks are often typical of water-borne epidemics and the true origin of the disease is thus often not identified.

As a consequence of the overall situation described above, the protection of large populations can no longer be assured by relying on past methods of standards setting. More direct cause and effect relationships between factor concentrations and adverse reactions must be established. This will permit the assessment of the degree of risk incurred by water users and will provide a basis for rational action by regulatory agencies. By setting RWQ standards on the basis of acceptable risk levels, rather than arbitrary factor concentrations, the water recreationist will be better protected.

The development of the cause-effect relationship into a concentration versus risk formulation is referred to in this report as the factor-to-criteria conversion. The first step required in the development of the conversion methodology was a detailed description and consideration of the known parameters of each factor involved in the study. The pertinent parameters were identified during the initial review of the available literature, but quantification of parameters could not be completed in the first phase of the program, in that much of the useful information is found in analogous studies throughout the literature or has to be reworked before being used as input for the mathematical analysis.

Health risks associated with primary contact use of recreational water include infections transmitted by pathogenic microorganisms; irritating, allergic, and debilitating responses to chemical and physical factors; and injuries sustained as a consequence of impaired visibility in turbid waters. Such risks are dependent on human, factorial, and aquatic parameters, interacting with the degree and type of exposure of the recreationist, as indicated in

Figure 1. A more detailed diagram (Figure 2) illustrates some of the potential recreational water/health hazard relationships, and portrays a complete view of the overall problem. The list of diseases included in the figure is based on literature citations for both drinking and recreation waters. Note that Figure 2 distinguishes the sources of contamination of the water as endogenous or exogenous. In the case of recreational water endogenous contamination, one assumes the water to have been safe prior to the entrance of recreationists. Exogenous pollutants include human, animal, industrial, and agricultural wastes, which enter the water as sewage or surface run-off.

Although Figure 2 presents an array of diseases potentially transmitted by the water route, relatively few of them are known to pose a serious threat to the aquatic recreationist. In general, the types of diseases whose transmission has been associated with primary body contact are (1) eye, ear, nose, and throat infections; (2) skin diseases; and (3) gastrointestinal disorders. Because most persons swallow only small amounts of water during swimming, and even less during other water-associated activities, enteric infection by recreational water is of much less significance than with drinking water. Nevertheless, cases of typhoid and other intestinal diseases, contracted from polluted water, have been reported in the literature, and there is growing concern that viral infections, particularly infectious hepatitis, may be contracted by bathing in sewage-polluted water (Camp, 1963).

This study represents an attempt to quantify the risks to the recreationist on the basis of data already available in the literature. Ideally, the needed criteria would be developed by exposing a user population to various concentrations of the factors of interest in a body of water. The incidence of adverse reactions would then be measured and the level of risk simply calculated. Such studies of course cannot be carried out. An approximation of a controlled exposure is a broad epidemiological study wherein the factor concentration in a water is measured and an accurate assessment is made of the disease incidence in the affected population over an extended period of time. Studies of this type have been attempted in the past, but they are of limited value for the purposes of this program. In the main, they have been designed with other than direct cause-effect relationships in mind, have been too limited in scope, or have not collected the type of information required for assessing risk versus factor concentration function.

Faced with this lack of directly pertinent data, the Envirogenics Co. undertook to develop a factor to criteria conversion methodology that could utilize and build upon the available data base. This study undertook to evaluate the data available on nine selected factors that, in each case, represented a varying spectrum of expected quantitative

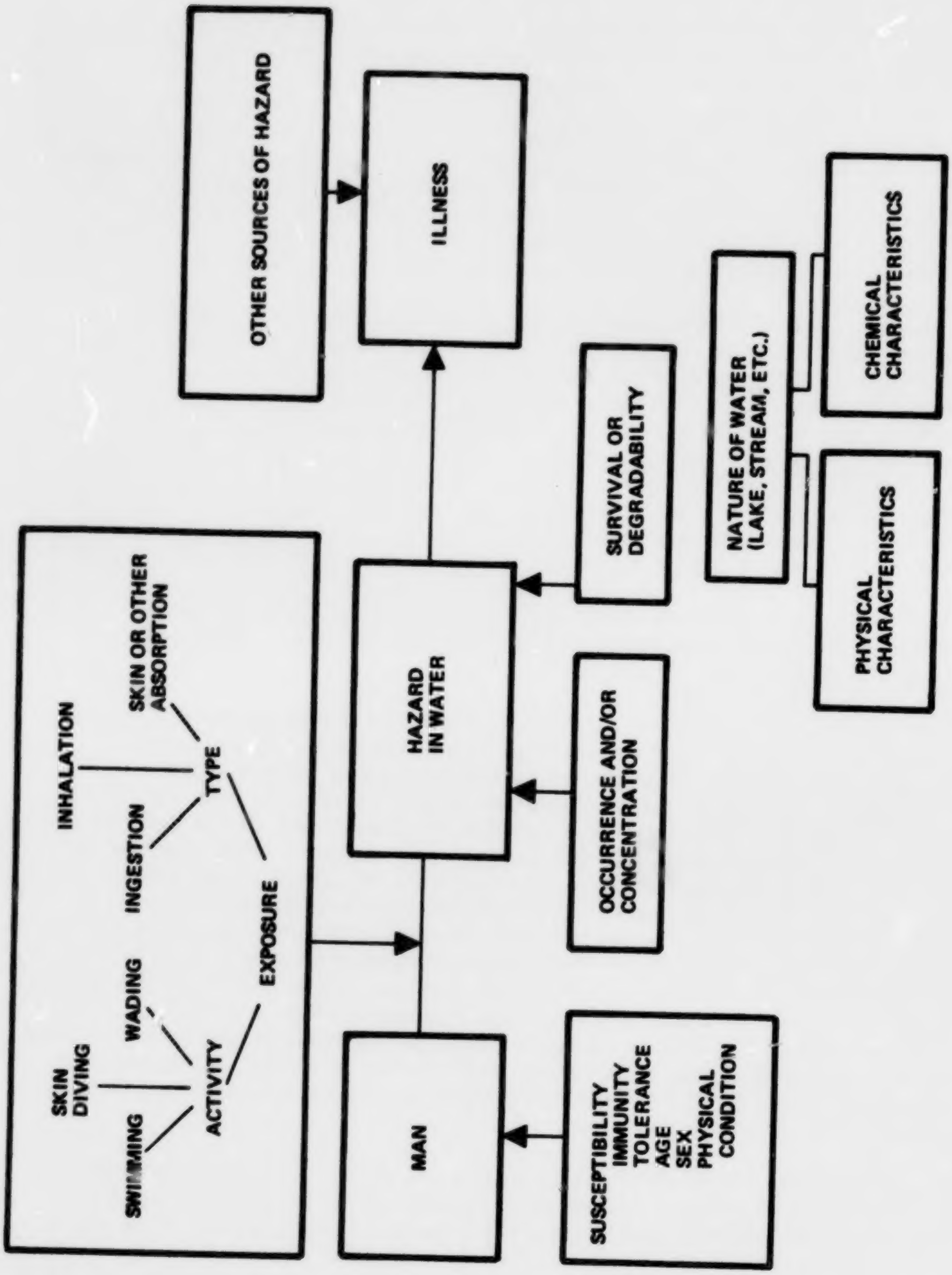
OTHER SOURCES OF HAZARD
ILLNESS
SWIMMING
SKIN DIVING
WADING
ACTIVITY
EXPOSURE
INGESTION
INHALATION
SKIN OR OTHER ABSORPTION
TYPE
MAN
HAZARD IN WATER
SURVIVAL OR DEGRADABILITY
OCCURRENCE AND/OR CONCENTRATION
SUSCEPTIBILITY
IMMUNITY
TOLERANCE
AGE
SEX
PHYSICAL CONDITION
NATURE OF WATER (LAKE, STREAM, ETC.)
CHEMICAL CHARACTERISTICS
PHYSICAL CHARACTERISTICS

Figure 1. Parameters of the Health/Hazard Relationship in Recreational Water

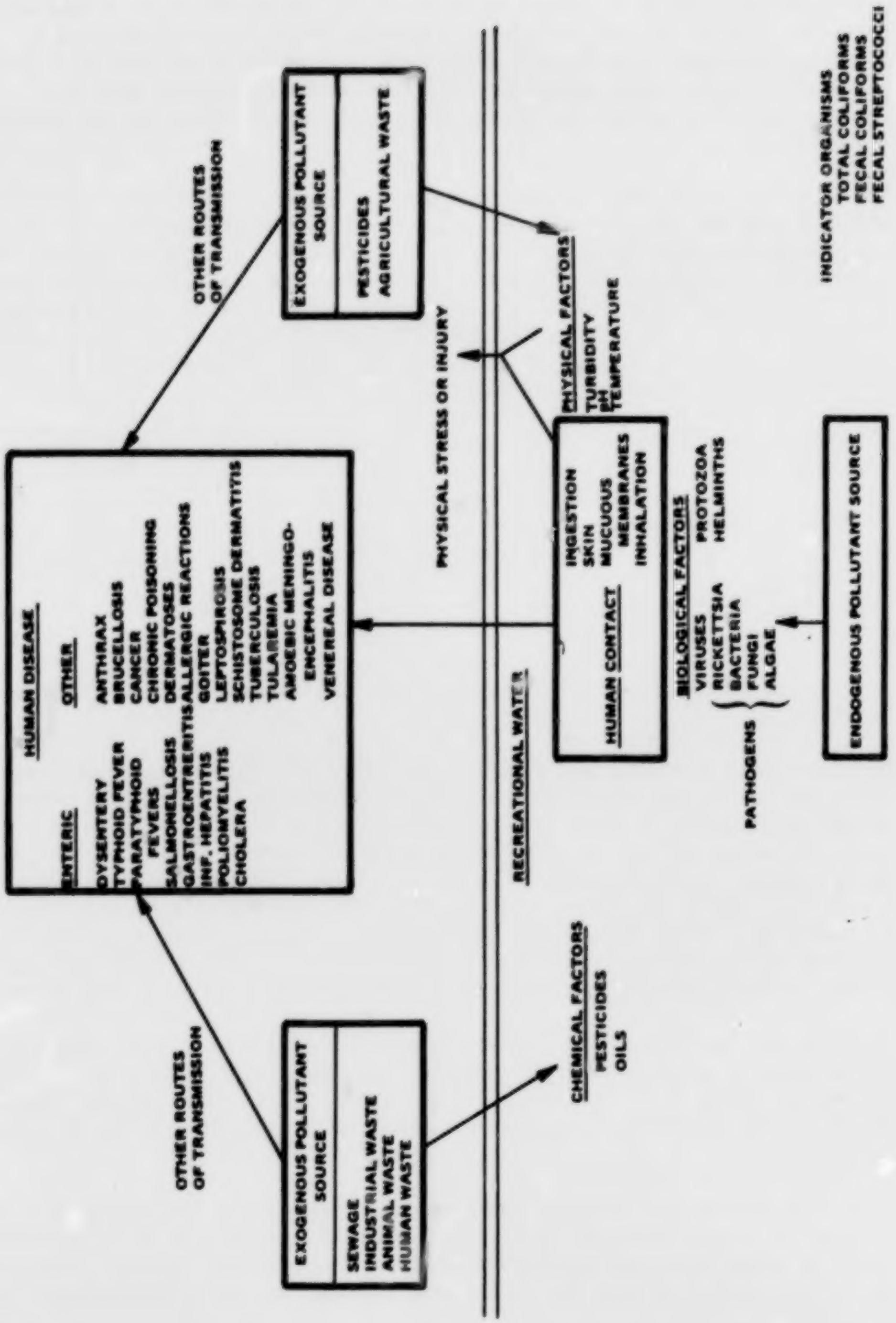
EXOGENOUS POLLUTANT SOURCE
SEWAGE
INDUSTRIAL WASTE
ANIMAL WASTE
HUMAN WASTE
OTHER ROUTES OF TRANSMISSION
HUMAN DISEASE
ENTERIC
DYSENTERY
TYPHOID FEVER
PARATYPHOID FEVERS
SALMONELLOSIS
GASTROENTERITIS
INF. HEPATITIS
POLIOMYELITIS
CHOLERA
OTHER
ANTHRAX
BRUCELLOSIS
CANCER
CHRONIC POISONING
DERMATOSES
ALLERGIC REACTIONS
GOITER
LEPTOSPIROSIS
SCHISTOSOME DERMATITIS
TUBERCULOSIS
TULAREMIA
AMOEBIC MENINGO-ENCEPHALITIS
VENEREAL DISEASE
EXOGENOUS POLLUTANT SOURCE
PESTICIDES
AGRICULTURAL WASTE
OTHER ROUTES OF TRANSMISSION
PHYSICAL STRESS OR INJURY
RECREATIONAL WATER
CHEMICAL FACTORS
PESTICIDES
OILS
HUMAN CONTACT
INGESTION
SKIN
MUCUOUS MEMBRANES
INHALATION
PHYSICAL FACTORS
TURBIDITY
pH
TEMPERATURE
BIOLOGICAL FACTORS
PATHOGENS
VIRUSES
RICKETTSIA
BACTERIA
FUNGI
ALGAE
PROTOZOA
HELMINTHS
ENDOGENOUS POLLUTANT SOURCE
INDICATOR ORGANISMS
TOTAL COLIFORMS
FECAL COLIFORMS
FECAL STREPTOCOCCI

Figure 2. Potential Recreational Water/Health Hazard Relationships

information. Data on these factors were collected from a variety of sources, all of it was reviewed, and much of it was abstracted. This gleaning process resulted in sufficient material to permit the development of a model that would use current dose response data for estimating risk in waters containing various amounts of the factors of interest.

Throughout the study, tentative models of the conversion methodology were designed, modified, and discarded in the attempt to accommodate the available information. This resulted in a shifting of emphasis to what was considered as a suitable data base; thus additional searching was sometimes required. This evolutionary type development cycle resulted in the selection of a methodology that could accept raw dose response data for a given factor, along with information on factor distribution, and output an assessment of risk.

A problem faced early in the program was the lack of sufficient data directly linking recreational use of water to disease. Thus a system had to be developed that utilized information generated in non-water related health programs. Experiments were available from such studies that related the dosage of a given factor to disease incidence in humans. This then determined how many people could be expected to become ill if they somehow acquired a stated dose of the factor, whether from water, food, or other transmission modes. There were also available a limited number of reports from several pollution monitoring agencies that presented the distribution of various factors in a water.

By considering these two inputs - one derived from medical research, the other from pollution monitoring - an estimate of risk to a recreationist using a given body of water could be made. This risk was based on calculating the frequency with which the recreationist would encounter a concentration of the factor sufficient to cause an adverse reaction. Once the basic risk is derived, the effects of selectively limiting access to the water can be determined. This series of truncations of the factor concentration results in a curve that relates factor concentration in a given water to probability of disease.

This model has been used to calculate the risks to a user population using waters containing Salmonella, viruses, and coliforms. The model is ready for testing in an epidemiological situation to determine whether the predicted incidence of ill effect coincides with observed effects.

It is suggested that regulatory agencies base their water quality standards for a given factor on a "level of acceptable risk" rather than on a specific concentration limitation. In that risk is dependent on both population susceptibility and concentration distribution, no single number could be universally applicable to all recreational waters. An agency responsible for meeting the RWQ standards that were set to protect a population would meet the requirements by determining the factor concentration in waters under their charge and, by application of the conversion methodology, construct a risk curve to set the appropriate numerical values to be monitored in each of their waters.

The factor-to-criteria conversion methodology incorporates several important features:

> The methodology requires relatively simple and understandable inputs: dose-response data and factor concentration data.
>
> Data processing is greatly simplified and rapid through the use of computer programming.
>
> Research needs and priorities can be established. The data requirements point out research deficiencies when information is collated.
>
> The conversion is completely objective.
>
> The model is amenable to revision and the acceptance of new inputs. Qualifying circumstances can be incorporated and evaluated.
>
> The model can be used as a predictive tool.
>
> A meaningful output is obtained. The factor-risk curve is immediately useful for setting RWQ standards.

With the successful completion of the initial program, the project should now be extended to exploit the features just discussed. The aim should be to make the methodology a dynamic instrument for protecting water quality and public health. The availability of the factor to criteria conversion methodology permits an objective estimate of risk for given hazard concentrations. As will be demonstrated in the following sections, merely carrying out the analysis on any given factor produces new insights into the significance of some of the published research. For the first time a working tool has been developed that has the potential of establishing water quality criteria on an objective and rational basis. Its acceptance by public health officials requires that confidence in the methodology be established by expanding its analytical capabilities and verifying its predictions.

SECTION IV

DATA BASE

LITERATURE COMPILATION

Meaningful mathematical analyses could not be initiated until certain conditions had been met. These conditions included the following:

Identification of the relevant parameters of the factors

Assembly of bibliographical references

Acquisition of the pertinent literature

Extraction of the relevant information contained therein

The parameters of importance for each of the nine selected factors were derived from a study of basic reference books, recent review articles, the periodical literature, and an overview of related subjects in abstract journals.

As a necessary pre-requisite to the incorporation of information into a mathematical model, a thorough literature search was conducted to retrieve relevant data for analysis. The emphasis was largely on the recent literature with a view toward ultimately retaining only data of informational value.

One of the major difficulties encountered in establishing direct cause-effect relationships between RWQ factors and human reaction (particularly disease) is the fact that many of the pathogenic agents of concern have alternate routes of transmission, and that most of the definitive studies on these pathogens are concerned with disease engendered by a route other than accidental water ingestion, inhalation, or absorption. Studies on waterborne disease and drinking water have limited application of the problem of the risk faced by the recreationist who imbibes relatively small amounts of water during water contact activities.

Nevertheless, such information may be quite useful (after extrapolation) in areas where minimal direct data is available with respect to disease transmission and recreational water quality. Certainly, many of the current RWQ standards and regulations have their origin in studies on drinking water quality, and these bases had to be considered carefully to determine their applicability to the present investigation.

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In order to attack the problem of a discriminating and systematic literature search early in the program, a tentative work plan was constructed as shown in the accompanying diagram (Figure 3).

The standard procedures of selecting descriptions, searching literature indexes and abstract journals, as well as re-examining the references cited in reviews, were followed in the initial phases of this study. From the diagram it can be seen that the process is a cyclical one, one that was intended to include gradually most, if not all, of the literature pertinent to the investigation.

It is estimated that approximately 20,000 relevant titles were scanned for selection of appropriate articles. When possible, initially selected titles were further screened by examination of abstracts before final selection for acquisition was made. The indexes and abstract journals used were Index Medicus, Chemical Abstracts, International Abstracts of Biological Science, Bibliography of Agriculture, and Readers Guide to Periodical Literature. As the bibliography was compiled, it was expanded by including the references appearing in articles as they were read and/or acquired.

From an original list of approximately 1000 selected titles, about 750 were read and abstracted. From the 750 articles, books, reports, proceedings, and other sources, approximately 350 abstracts were chosen for reference and inclusion in the annotated bibliography. It is believed that most of the pertinent and key literature retrievable by feasible search methods and descriptor usage has been identified, either in the reference material, or secondarily through its references, as far as the basic purpose of the investigation is concerned. Considerable useful material may still be retrievable from the analogous literature.

In addition to the manual methods of literature retrieval used in this study, the National Library of Medicine was asked to use its MEDLARS system to retrieve titles relative to the present task. It should be noted that the MEDLARS file gives access to titles indexed only after 1963. With the use of the descriptors supplied, a computer run was made on all available literature (estimated to be about 750,000 titles). From this run, 87 references were received, of which only 20 proved useful. However, in the specific area of toxicity of pesticides to man, some 300 titles were retrieved.

HISTORICAL BASIS OF STATE RECREATIONAL WATER QUALITY CRITERIA AND STANDARDS

The Federal and State Role in Water Pollution Control

The present survey was conducted to review the water criteria and

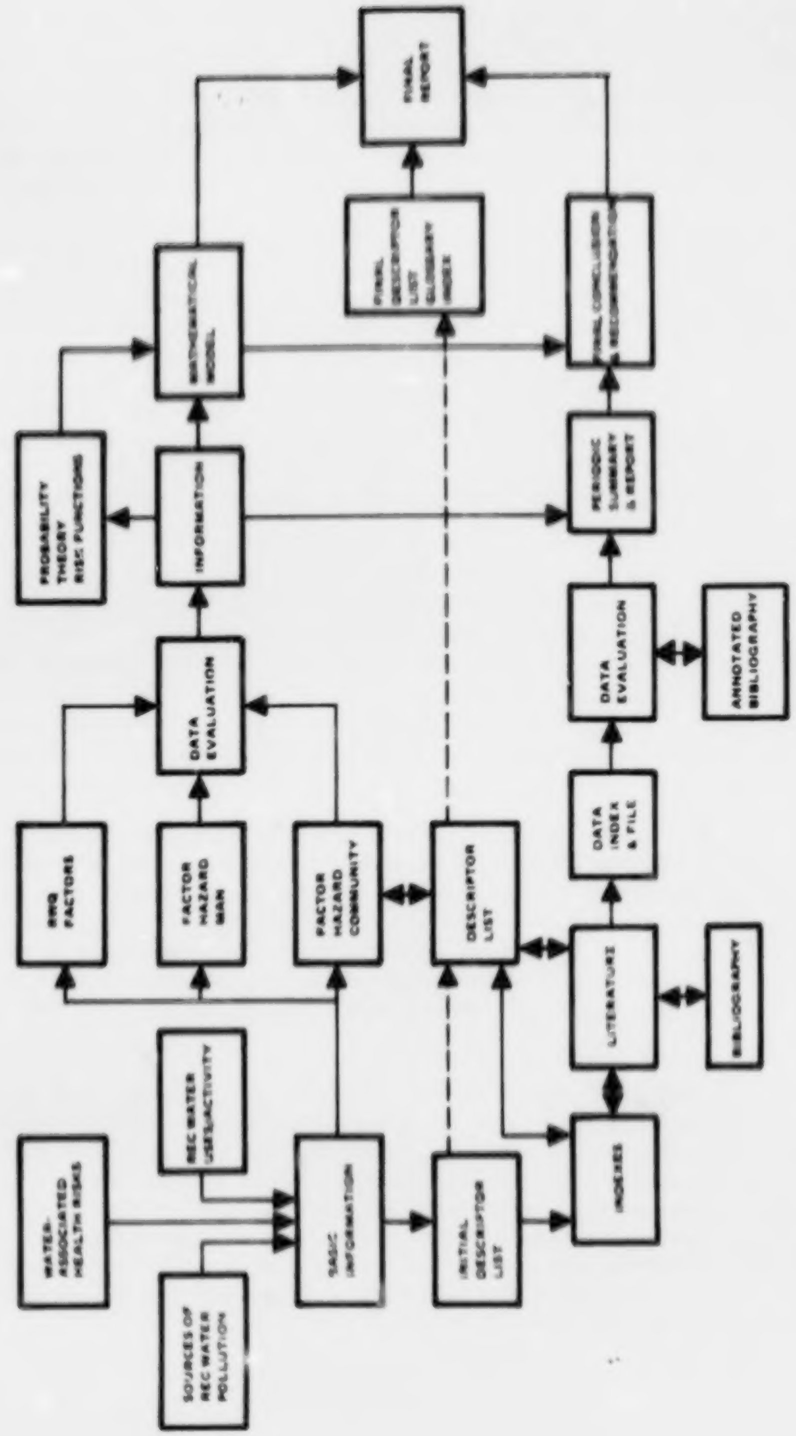

SOURCES OF REC WATER POLLUTION
WATER-ASSOCIATED HEALTH RISKS
REC WATER USE/ACTIVITY
BASIC INFORMATION
INITIAL DESCRIPTOR LIST
INDEXES
LITERATURE
BIBLIOGRAPHY
DESCRIPTOR LIST
RISK FACTORS
FACTOR HAZARD MAN
FACTOR HAZARD COMMUNITY
DATA INDEX & FILE
DATA EVALUATION
ANNOTATED BIBLIOGRAPHY
DATA EVALUATION
INFORMATION
PROBABILITY THEORY RISK FUNCTIONS
PERIODIC SUMMARY & REPORT
MATHEMATICAL MODEL
FINAL DESCRIPTOR LIST GLOSSARY INDEX
FINAL CONCLUSION & RECOMMENDATIONS
FINAL REPORT

Figure 3. General Investigation Plan

standards set by the various states and territories for primary contact situations in recreational waters. The objective of the analysis was to investigate the parameters of total coliforms, fecal coliforms, pH, temperature, clarity, pesticides, and oils, and, most importantly, to determine the rationale for the standards adopted. This aspect of the investigation was conducted by Montgomery Research, Inc., Pasadena, California, acting as subcontractor on the program.

In 1948, the first Federal Water Pollution Control Act was passed as Public Law 845, 80th Congress, although the Federal role in water pollution control had been defined earlier in three acts - the Rivers and Harbors Act of 1899, the Public Health Service Act of 1912, and the Oil Pollution Act of 1924. The 1948 Act provided for water pollution control in the Public Health Service, as follows: "In the development of such comprehensive programs due regard shall be given to the improvements which are necessary to conserve such waters for public water supply, propagation of fish and aquatic life, recreational purposes, and agriculture, industrial and other legitimate uses."

Comprehensive water pollution control legislation was enacted by the 84th Congress, and was signed into law on July 9, 1956, as the Federal Water Pollution Control Act, Public Law 660. This Act extended and strengthened its 1948 precursor, which expired on June 30, 1956. Further amendments to the Act in 1961 improved and strengthened it by extending Federal authority to enforce abatement of pollution in intrastate as well as interstate or navigable waters.

The Federal Water Pollution Control Act (PL 660) was further amended by the Water Quality Act of 1965 (PL 89-234), which declared that it is "the policy of Congress to recognize, preserve and protect the primary responsibilities and rights of the States in preventing and controlling water pollution, to support and aid technical research relating to the prevention and control of water pollution, and to provide Federal technical services and financial aid... in connection with these activities."

The 1965 Act requires that "comprehensive programs shall be developed for eliminating or reducing the pollution of interstate waters and tributaries thereof," and retains the same wording as quoted above from the Act of 1948 in connection with comprehensive programming. Research, investigations, experiments, demonstrations, and studies relating to the causes, control, and prevention of water pollution of interstate or navigable waters in or adjacent to any state or states which endangers the health and welfare of any persons, are provided for in the Act.

To attain these ends by appropriate state legislation, the 1965 Act specified that each state file a letter of intent and, after public hearings and before June 30, 1967, adopt water quality criteria and a plan for applying them to interstate waters within the state. Such criteria and plan, if consistent with the Act, thereafter would be the applicable interstate water quality standards. In establishing such standards, consideration was required to be given to their use and value for public water supplies, propagation of fish and wildlife, recreational purposes, and agriculture, industrial, and other legitimate uses (Mackenthun and Ingram, 1967).

The following account describes the results of state compliance with the provisions of the Act with respect to waters intended for recreational use, and compares the criteria and standards developed by the various jurisdictions.

Method of Conducting Survey

To investigate current state standards for recreational waters, a letter of inquiry was sent to fifty states and eight territories and districts on July 10, 1969. This letter is reproduced in Appendix A. A second letter was sent October 14, 1969, to those states not responding; telephone calls were also made in some instances. At the completion of the study, replies had been received from fifty states and six districts or territories. The various jurisdictions forwarded copies of their published water quality documents for review. The information developed from these documents is presented in Table 40 of Appendix A.

SUMMARY AND DISCUSSION OF CURRENT STATE STANDARDS

The rationale for the various state standards is compiled in Table 1. Twenty-six states and territories gave no information regarding the basis for their standards. However, it is assumed that all states conducted public hearings in accordance with the State Administrative Procedure Act. Nine states base their criteria wholly or in part on testimony and reports received at the public hearings. Eleven states followed recommendations by the FWPCA in establishing standards.

South Dakota's water quality criteria were developed by contract between the South Dakota School of Mines and Technology and the South Dakota Department of Health. Five factors formed the framework within which the standards were constructed: important pollutants, natural water quality, existing and potential beneficial uses, enforcement problems, and Federal requirements (Barker, 1969). Extensive studies were conducted before South Dakota's standards were adopted.

TABLE 1

Rationale for state standards

Basis	State or Territory
Review of testimony and reports given at public hearings	Alaska, Idaho, Iowa, Missouri, Nebraska, North Carolina, North Dakota, Ohio River Valley, Oklahoma
Recommendations of FWPCA	Colorado, Connecticut, Delaware, District of Columbia, Nevada, North Dakota, Pennsylvania, South Carolina, Texas
Consideration of present conditions, contributing factors, and quality requirements for specified uses	Arizona, Oklahoma
Specialized study conducted by state in conjunction with FWPCA	Georgia, South Dakota
Literature review, present scientific knowledge, experience and judgment	Alabama, Hawaii, Michigan, South Carolina, South Dakota, Virginia, Washington, Wisconsin
Consultation with neighboring states	Colorado, Ohio River Valley, South Dakota
Recognition of criteria of other states or agencies	Kentucky (ORSANCO criteria), Massachusetts (New England State Water Pollution Control Commission)
Good sanitary engineering practice and consultation with the affected parties	Mississippi
Past experience	Nebraska, Texas, Washington
Present and potential uses, and existing water quality as indicated by monitoring programs	Louisiana, Wyoming

TABLE 1

Rationale for state standards

(Continued)

Basis	State or Territory
Professional knowledge of the board's staff and judgment of the board	Texas, Wisconsin
Not given	Arkansas, California, Delaware River Basin, Florida, Guam, Illinois, Indiana, Kansas, Maine, Maryland, Minnesota, Montana, New Hampshire, New Jersey, New Mexico, New York, Ohio, Oregon, Puerto Rico, Rhode Island, Tennessee, Utah, Vermont, Virgin Islands, West Virginia

Two states adopted the criteria of another agency for their own state use. Kentucky accepted the Ohio River Sanitary Commission criteria, and Massachusetts adopted the criteria of the New England State Water Pollution Control Commission.

The Table 1 summary illustrates the very general character of the bases used in developing the state RWQ criteria. The survey did not elicit any epidemiological data, direct cause and effect relationships, or statistical studies to support the proposed limits. In some cases, bibliographies were contained in the documents, but specific reference to their use in formulating the criteria was not indicated.

Examination of the data on primary contact recreational total coliform standards shows that the most commonly accepted standard is 1000/100 ml, with one state being as restrictive as 50/100 ml, and others as liberal as 5000/100 ml. These standards imply that an acceptable risk for recreational waters has been assumed by the regulatory agencies. The rationale for the 1000/100 ml figure is apparently based on field epidemiological analysis and studies along the Ohio River, Lake Michigan, and Long Island Sound as reported by Stevenson (1953), and comparisons of die off rates for coliforms and enteric pathogens made by Kehr and Butterfield (1943). Of particular interest here is the analysis of Streeter (1951), in which he used the data of Kehr and Butterfield to develop a procedure for calculating the risk of typhoid infection for bathers in the Ohio River. Assuming a total coliform count of 1000 coliforms per 100 ml, he determined the chance of contracting typhoid from swimming daily for 90 days would be 1 in 950. Turning to the extremes represented by current state standards, namely, 50/100 ml to 5000/100 ml, application of the same methodology and conditions reveals a typhoid risk range of 1/19,000 to 1/150, which expresses more clearly the implication of the coliform standards. However, the studies of Moore (1959) on the health risks in sewage-contaminated seawater indicated that the incidence of illness among swimmers was much less than predicted from Streeter's work. This discrepancy needs to be resolved. It may be that the use of the fecal coliform as an indicator would be more predictive of risk than the total coliforms used by Streeter and earlier workers.

Total Coliforms

Total coliform standards have been adopted by 35 states and territories. The standards range from 5000/100 ml in Illinois and Virginia (secondary contact), to 50/100 ml for Utah (see Table 2). Between the levels of 50/100 ml and 5000/100 ml lies an intermediate zone, with the majority of states adopting a standard of 1000/100 ml. Eight of the states with a total coliform standard also have a fecal coliform standard. These states are Colorado, Hawaii, Idaho, Iowa,

TABLE 2

Summary of total coliform standards for bathing waters

Total Coliforms (Not to Exceed)	State or Territory
5000/100 ml	Illinois, Virginia (secondary contact)
2400/100 ml	New York (boundary waters), Virginia (primary contact)
1600/100 ml	Louisiana
1000/100 ml	Alaska, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Indiana, Massachusetts, Michigan, Minnesota, Montana, Nevada (for Colorado River), North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Puerto Rico, Rhode Island, South Dakota, West Virginia, Wisconsin
500/100 ml	Vermont
240/100 ml	Idaho, New Hampshire
200/100 ml	Arkansas
50/100 ml	Utah

Michigan, Pennsylvania, Virginia, and Oklahoma.

Fecal Coliforms

Fecal coliform standards have been adopted by twenty-five states and territories. The range is from 1000/100 ml for Mississippi, Tennessee, Alabama, Michigan (partial body contact), and Georgia to 70/100 ml for the Virgin Islands (see Table 3). The Virgin Islands' standard is for coastal waters and is the standard for shellfish areas. The Wyoming fecal coliform standard specifies organisms not to exceed a 95-percent confidence limit of the historical data. The intermediate zone for the fecal coliform range is 200/100 ml.

There appears to be a trend toward the adoption of fecal coliforms as the preferred method in determining safety of bathing waters. Delaware measures fecal coliforms and fecal streptococci in addition to total coliforms. The Water and Air Resources Commission of Delaware "feels that fecal coliforms are a better indicator of pollution than total coliforms" (Vasuki, 1969). The state of Georgia has recently completed a study for FWQA concerning fecal coliform standards and will probably amend its standards as a result of the study. Kentucky, which recognized the ORSANCO criteria as the basis for their standards, has recently accepted the adoption of a fecal coliform standard. This action was encouraged by the FWQA (Martin, 1969). The ORSANCO water quality criteria were revised on May 15, 1969, to include a fecal coliform standard. Pennsylvania is the process of changing to a fecal coliform criterion for bathing waters. The standard being considered is similar to that recommended by the FWQA (Bordman, 1960).

pH

The range of pH standards is illustrated in Table 4. There are eleven states with a minimum pH of 6.0 for bathing waters. Those states are Alabama, Arkansas, Delaware River Basin, District of Columbia, Georgia, North Carolina, Louisiana, Mississippi, South Carolina, Tennessee, and West Virginia. Three states have a lower pH standard specifically set for swamp waters. Georgia has a pH limit of 4.5 for swamp waters, North Carolina 4.3, and South Carolina 5.0. There are two states (Louisiana and Montana) with a maximum pH standard of 9.5. Within this range of values, the majority of states are generally not set with recreational water usage as the prime consideration, but rather in consideration of the other uses such as propagation of fish life and domestic water supply.

Temperature

Waters are classified for multiple uses. The temperature of a

TABLE 3

Summary of fecal coliform standards for bathing waters

Fecal Coliforms (Not to Exceed)	State or Territory
1000/100 ml	Alabama, Georgia, Michigan (partial body contact), Mississippi, Tennessee
240/100 ml	District of Columbia, Maryland
200/100 ml	Arizona, Delaware River Basin, Guam, Hawaii, Illinois, Missouri, Nebraska, New Mexico, North Carolina, Ohio River Valley, Oklahoma, Pennsylvania, South Carolina, Tennessee (organized camps), Texas
100/100 ml	Colorado, Michigan
70/100 ml	Virgin Islands
	Fecal coliform standard for Wyoming not to exceed a 95% confidence limit of the historical data.

TABLE 4

Summary of pH standards

pH Range	Number of States
6.0 - 8.0	1
6.0 - 8.5	7
6.0 - 9.0	2
6.0 - 9.5	1
6.5 - 8.0	4
6.5 - 8.5	14
6.5 - 8.6	1
6.5 - 9.0	2
6.5 - 9.5	1
6.8 - 9.0	1
7.0 - 8.3	1
7.0 - 8.5	3
No reduction - 8.0	1

Number of states with minimum pH of 6.0	11
Number of states with minimum pH of 6.5	23
Number of states with minimum pH of 6.8	1
Number of states with minimum pH of 7.0	4
Number of states with maximum pH of 8.0	6
Number of states with maximum pH of 8.5	25
Number of states with maximum pH of 9.0	5
Number of states with maximum pH of 9.5	2

water is considered more critical to domestic, industrial, and commercial supply and to fish life than to primary contact recreational use. Temperature standards are, therefore, often set on the basis of suitability for fish, with the same standards applied to recreational waters. The temperature standards that have been set are quite varied as is shown in Tables 5 and 6. The top of the scale for maximum temperature permitted due to effluents is 96.8°F (Louisiana) and 57.2°F as the lowest (Hawaii). Fluctuations permitted above ambient but below maximum are shown in Table 6 and range from 10.0°F (Mississippi, Nebraska (summer maximum) and Georgia), to 1.5°F (Hawaii).

Pesticides and Other Toxic Substances

Forty-eight state and territory standards contain the phrase "Substances in concentrations or combinations which are harmful to human, animal, plant, or aquatic life are not permitted," or its equivalent. Pesticides are covered by this non-quantitative statement. This phrase relieves water quality administrators of the problem of dealing with a multiplicity of parameters. The lack of sufficient data on adverse effects of materials raises questions about threshold effect levels and makes establishment of standards difficult. Table 7 summarize these standards. Two states do have quantitative standards for toxic materials. Arkansas states the level of toxic substances shall not exceed 0.1 of the 48-hour median tolerance level (Anon, 1967, Arkansas). South Dakota's standard reads "no concentrations greater than 0.1 times the acute (96 hr) median lethal dose for short residual compounds, or 0.01 times the acute median lethal dose for accumulative substances" (Anon, 1967, South Dakota). Six states have no standard set at the time of this survey (Rhode Island, Florida, Pennsylvania, Virginia, Wisconsin, and Wyoming). Wyoming states in their criteria literature that specific limits will not be established for toxic materials because all possible compounds, combinations, and effects are not known (Anon, 1968, Wyoming). The future will very likely see the formalization of standards in this area for many states as knowledge of toxic substances increases.

Clarity

Thirty-eight states have non-quantitative statements concerning clarity such as permitting no increase in turbidity that would impair any usages specifically adopted for the water in question (see Table 8). The standards that have been adopted range from a high of 50 Jackson Units (JU) - Arizona warm water streams - to 5 JU - Idaho and Oregon. Some states specify Secchi disc clarity in their standards (Hawaii, Nebraska, Guam, and Tennessee). Arizona, Delaware River Basin, Missouri, Montana, Idaho, and Oregon use a Jackson turbidity unit standard. Minnesota specifies a limit of 10, but does not indicate whether reference is to JU or Secchi disc clarity.

TABLE 5

Summary of Maximum Temperature Standards

Maximum Temperature, °F (due to effluents)	State or Territory
96.8	Louisiana
95.0	North Carolina
93.2	South Carolina, Georgia
93.0	Arizona, Iowa (summer maximum), Kentucky (summer maximum), Mississippi, Tennessee
90.0	Alabama, District of Columbia, Michigan, Missouri, New York (non-trout waters), North Dakota, Virgin Islands
87.0	West Virginia (summer maximum)
86.0	Delaware River Basin
85.0	Connecticut, Guam
73.0	Iowa (winter maximum), Kentucky (winter maximum), West Virginia (winter maximum)
70.0	New York (trout waters)
64.4	Nevada (summer maximum, Colorado River above Davis Dam)
57.2	Nevada (winter maximum, Colorado River above Davis Dam)
Non-quantitative statements such as: waters shall be so protected against controllable pollution, including heat, as to be suitable at all times for usage under the waters classification.	Alaska, Maryland, Massachusetts, New Hampshire, New Jersey, Utah, Vermont, Washington
No standard set solely for recreational water protection.	Arkansas, California, Colorado, Florida, Illinois, Indiana, Kansas, Maine, Minnesota, Montana, New Mexico, New York, Ohio, Ohio River Valley, Pennsylvania, Puerto Rico, Rhode Island, South Dakota, Texas, Virginia, Wisconsin, and Wyoming

TABLE 6

Summary of Temperature Fluctuation Standards

Fluctuations Permitted Above Ambient but Below Maximum, °F	State or Territory
10.0	Georgia, Mississippi, Nebraska (summer maximum)
7.0	North Carolina
5.4	Louisiana
5.0	Delaware, Delaware River Basin, District of Columbia, Missouri, Nebraska (winter maximum), New York, Oklahoma
4.0	Connecticut
2.0	Idaho (when stream temperatures are 64°F and below), New York (summer in trout waters), Oregon
1.5	Hawaii
	Alabama standard specified no more than 10% increase in temperature due to addition of waste

TABLE 7

Summary of Pesticide and Oil Standards

Standard	Number of States
No standard set	6
Non-quantitative - statements such as no toxic materials in concentrations harmful to human or wildlife.	48
Level of toxic substances shall not exceed 0. 1 of 48 hour median tolerance level.	1 (Arkansas)
No concentrations greater than 0. 1 times the acute (96 hr) median lethal dose for short residual compounds, or 0. 01 times the acute median lethal dose for accumulative substances.	1 (South Dakota)

TABLE 8

Summary of Clarity Standards

Standard	State or Territory
50 JU, warm water streams 10 JU, cold water streams 25 JU, warm water lakes 10 JU, cold water lakes	Arizona
Maximum monthly mean 40 units; Maximum 150 units.	Delaware River Basin
Secchi disc determination shall not be altered from natural condition more than 10%.	Guam, Hawaii, Nebraska
Shall not exceed 20 turbidity units due to effluents.	Missouri
Shall not exceed 10 turbidity units due to effluents.	Minnesota, Montana
Shall not exceed 5 turbidity units above natural value.	Idaho and Oregon
Secchi disc clarity of 5 feet at all times.	Tennessee
No standard given.	Virginia
Non-quantitative statements such as no allowable increase in such concentrations that would impair any usages specifically assigned to the waters classification.	38 States or Jurisdictions

SUMMARY

It is apparent that considerable variability still exists among the standards promulgated for the various states, despite Federal efforts to achieve uniformity. However, differing local conditions (physical, political, and economic) also play a role in setting standards. The concern here, however, is to discover the specific data that has served as the scientific basis for the standards. In general, it is apparent that additional source information is needed if the basis of the standards are to be analyzed in depth. Further investigation along these lines should be pursued in future work, as an adjunct to the search for current epidemiological data in selected areas. Although intensive pursuit of the data base may not produce significantly useful results in most of the areas of concern from the standpoint of cause-effect analyses, additional efforts to accumulate background information from the various health departments would permit a more detailed interpretation of criteria rationale than the general information now in hand.

SECTION V

THE FACTOR TO CRITERIA CONVERSION METHODOLOGY

The central objective of this program has been the development of a procedure by which information on a given water quality factor can be converted into criteria. Once this conversion is carried out for various concentrations of the factor of interest in a water, the degree of danger to a recreationist entering that water can be stated as the likelihood of infection or harm. A regulatory agency could then use the criteria to set standards based on the concentration of the factor in the water that produces a level of risk deemed acceptable by the agency.

RATIONALE FOR THE DEVELOPMENT OF THE MODEL

As indicated in Section IV, water quality criteria have been established based upon, among other things, the past experience of an investigator, epidemiological information, and even logical assumptions or carryovers from other types of work. As can be recognized, these methods are rather subjective and depend upon the expertise of various individuals.

A more desirable approach is to develop a method of establishing criteria by which the risk to an individual or population can be assessed on a purely objective basis. This can be done by determining the response of individuals to different quantities of the factor in question. By exposing a large number of individuals to the factor, a dose response curve can be prepared, based upon the probability that a given dose of the factor will produce an adverse effect. This in fact represents a statement of the "capability" of the recreationist to withstand a given challenge dose, and represents the first of the two inputs necessary to determine the risk to the recreationist.

In order to fully assess the risk an individual faces when entering a given water, it is also necessary to know the likelihood that he will encounter a given dose of the factor in the water. By knowing how the factor is distributed through the water and the frequency of occurrence of various concentrations of that factor, the probability distribution for the factor in the water can be estimated. The risk faced by the recreationist is determined by computing the probability that the recreationist will encounter a concentration of the factor that will likely result in a given adverse response.

DESCRIPTION OF THE MODEL

A mathematical model was developed that incorporates, on a quantitative basis, the features discussed above. The dose-response data, which represents the relationship between the amount of the factor ingested and the number of people subsequently infected, are converted into a probability density function. This requires that the type of distribution of the sampled population be determined. This probability distribution has been designated as the capabilities vector, C_p.

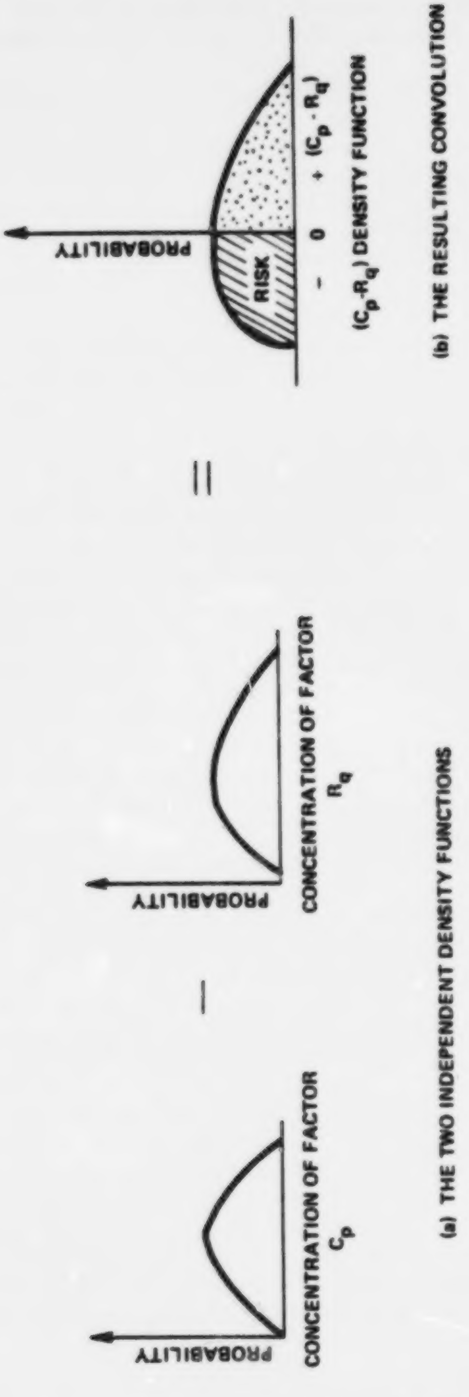
A second probability distribution is determined that describes the likelihood that a recreationist would encounter a given concentration of the factor when he enters a given body of water. This represents the requirement placed on the recreationist and has been designated as the requirements vector, R_q. These two probability distributions are represented in Figure 4a. They make up the fundamental information that is required as input into the model.

The risk faced by the recreationist can be expressed as the interaction of the two distributions. This risk can be defined as the probability that the water's requirement on the recreationist exceeds that individual's capability to withstand that challenge dose. The probability of risk for a given concentration of the factor is obtained by computing the probability that R_q (factor concentration in the water) is greater than C_p (concentration of factor producing adverse response). This is obtained by deriving the probability density function of the difference ($C_p - R_q$) and then computing the probability that ($C_p - R_q$) is negative or that the requirement exceeds the capability. The risk that the concentration of the factor in this water (R_q) will exceed the harmful dose (C_p) and hence cause an adverse effect is the area under that portion of the probability density function that is to the left of zero (Figure 4b). This convolution (subtraction) results in the basic risk probability function describing the likelihood of infection faced by an individual entering a given water. This risk can be lessened by making certain decisions on limiting expose of the population by various alternatives.

ANALYTICAL IMPLEMENTATION

It is apparent that the form of the distribution of the dose-response data and the factor-concentration data together determine the probability distribution of the convolution. As a consequence, it is critical that the frequency distribution of the sampled population be closely approximated. There are several commonly used methods for fitting a particular density function to a sample (Korn & Korn, 1968). The "Method of Maximum Likelihood," which often involves complicated computations, is the more accurate of those readily adaptable to this program. This method has been chosen to fit the data available on the factors of interest to this program to five possible statistical distributions: (1) normal, (2) exponential, (3) gamma, (4) Weibull, and (5) lognormal.

Once these distributions have been developed for the data at hand, it is necessary to test whether the assumptions made about each distribution are reasonable. That is, which sample distribution most closely approximates the true population distribution? The Kolmogorov-Smirnov or "d-test" for goodness of fit is one of many tests designed for this purpose (von Alven, 1964). By applying this test, the most probable distribution for the data at hand is selected from among the five choices.



PROBABILITY
CONCENTRATION OF FACTOR
C_p
−
PROBABILITY
CONCENTRATION OF FACTOR
R_q
=
PROBABILITY
RISK
− 0 + $(C_p - R_q)$
$(C_p - R_q)$ DENSITY FUNCTION

(a) THE TWO INDEPENDENT DENSITY FUNCTIONS

(b) THE RESULTING CONVOLUTION

Figure 4. The Factor to Criteria Conversion

An existing computer program, entitled BSTFIT by the author, is available that can perform a combination of fitting the input data into all five possible distributions, and then performing the Kolmogorov-Smirnov analysis to select the most likely fit (Dudley, 1970).

RISK AS A FUNCTION OF CHANGING REQUIREMENTS

Once a convolution has been carried out, the probability of infection or adverse reaction of a population entering that water can be stated. However, it is important to know how much this risk to the population would be reduced if entry to the water was banned whenever the factor concentration exceeded a given level. By closing a water at these times, the maximum exposure of a population is reduced and as a consequence the risk should also be reduced. The setting of such maximum limits truncates the factor concentration probability function (R_q distribution) and results in a modified or enchanced convolution (Figure 5).

Regardless of where the factor concentration curve is truncated, the area under the curve has to remain constant. The total area under the curve after the convolution is carried out also remains constant, but the shape of this latter curve changes. This results in a tendency of the curves to shift to the right of zero, resulting in a smaller area to the left or risk portion of the curve. By selectively truncating the factor concentration distribution - by the setting of stricter and stricter limits - a series of convolutions is obtained, each with a smaller risk area. The area of each risk curve can then be plotted as risk versus factor concentration and the confidence limits determined. The factor to criteria conversion is now complete. The equation for the resultant curve is then derived. The resultant polynomial of best fit is subjected to an analysis of variance and the confidence limits at the desired level of significance determined. This can then be presented as a plot of risk vs factor concentration and used for selecting appropriate standards.

This process is illustrated in Figure 5 where the basic Rq curve is truncated at the mean plus 2 standard deviations ($\mu + 2\sigma$) and at the mean plus 1 standard deviation ($\mu + 1\sigma$). A truncation at the mean would require that the water be denied to users 50% of the time, a highly undesirable situation. The resultant series of convolutions results in successively decreasing the risk portion of each convolution.

In the determination of risk, a computer program, RADOP, was available for performing these calculations. This program simultaneously carried out the series of successive truncations and the corresponding convolutions, along with the confidence limits. A third program was developed to fit the data to various polynomials and this was used to determine the type of equation that best describes the criteria, along with the confidence limits. A schematic of the computer implementation of the entire methodology just discussed is presented in Figure 6.

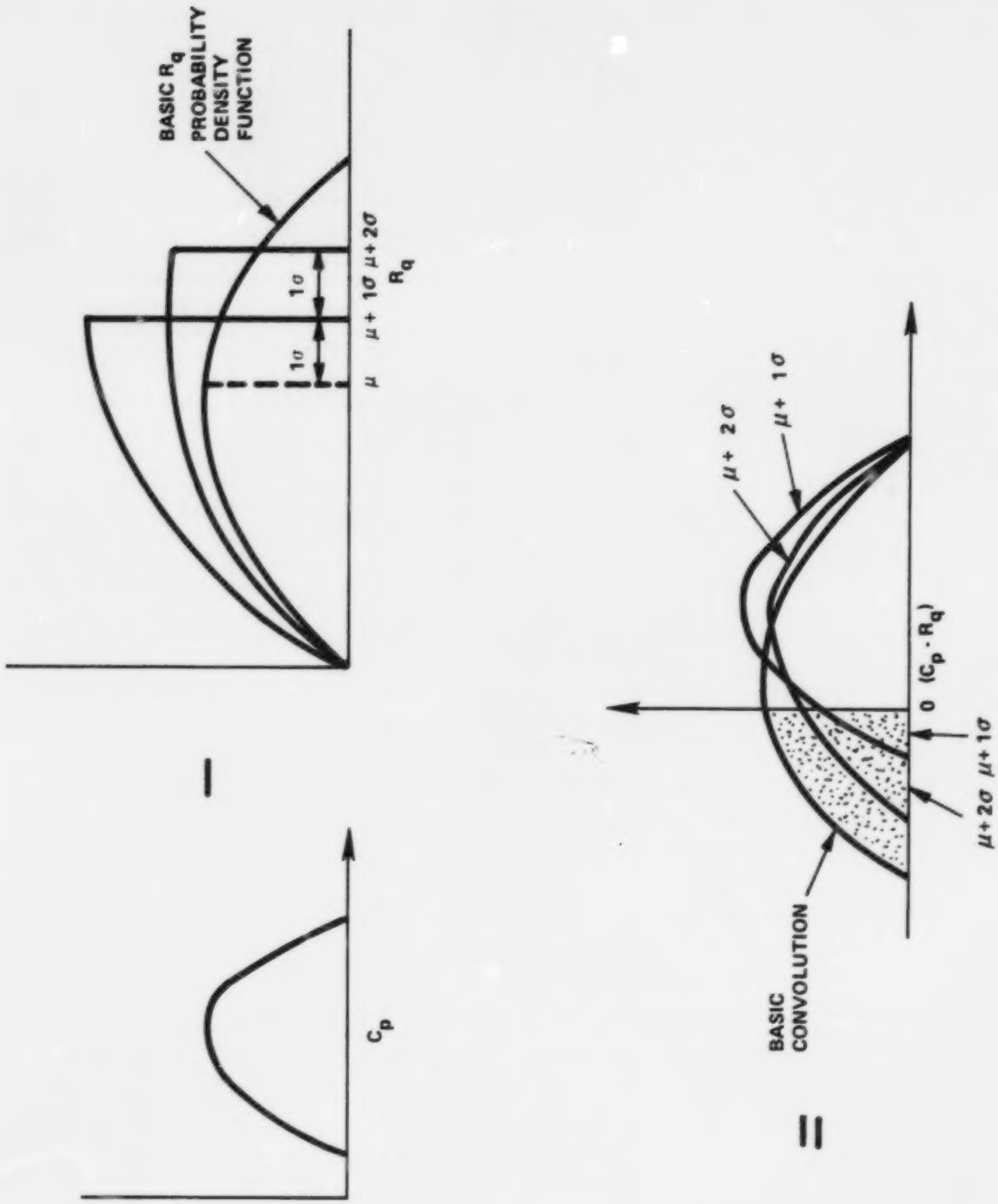

BASIC R_q PROBABILITY DENSITY FUNCTION
1σ
1σ
μ
μ + 1σ
μ + 2σ
R_q
I
C_p
μ + 2σ
μ + 1σ
0 $(C_p \cdot R_q)$
μ+2σ μ+1σ
BASIC CONVOLUTION
II

Figure 5. The Truncated and Enhanced Convolution Series

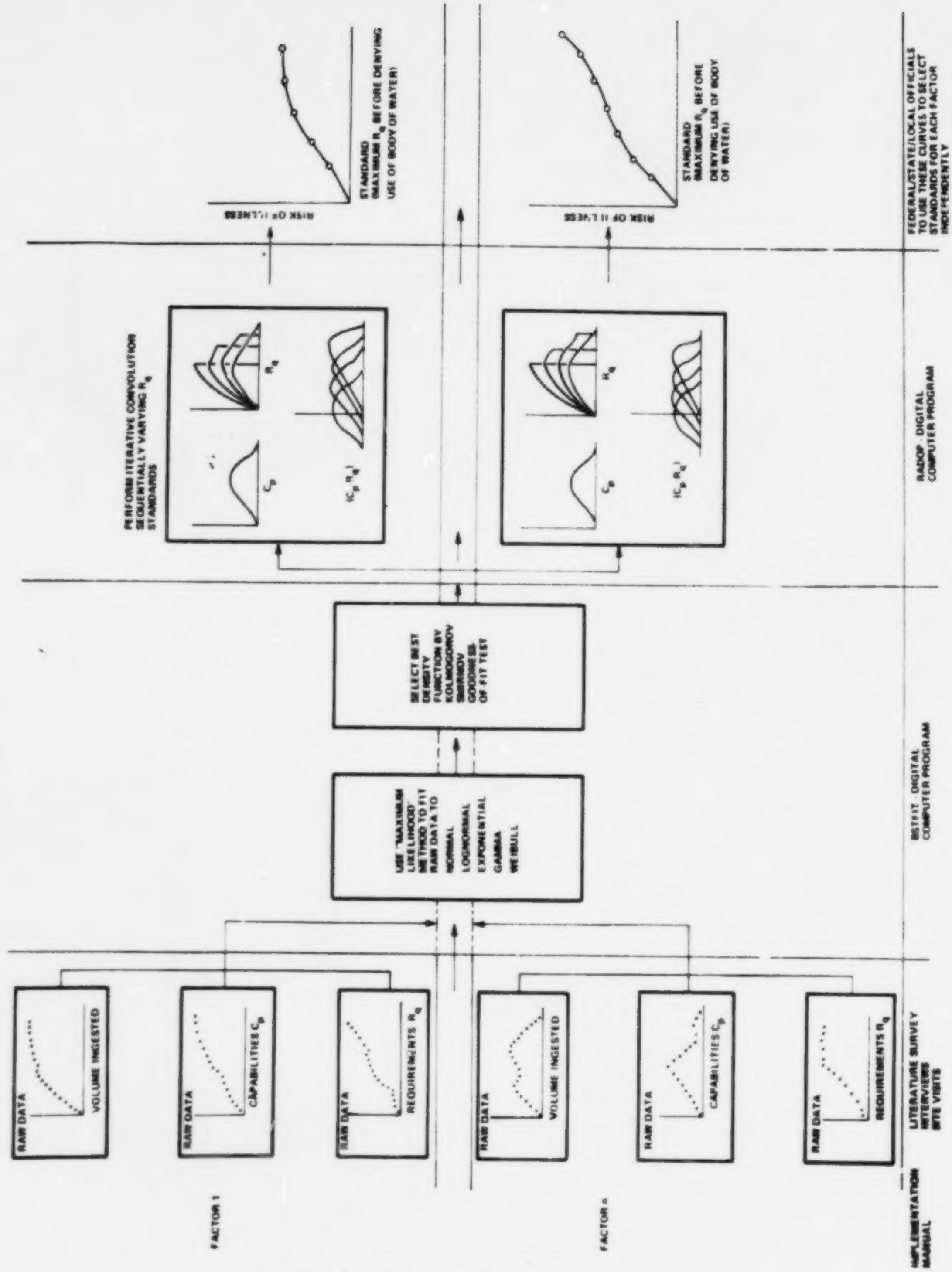

FACTOR 1
FACTOR n
RAW DATA
VOLUME INGESTED
RAW DATA
CAPABILITIES C_p
RAW DATA
REQUIREMENTS R_q
USE "MAXIMUM LIKELIHOOD" METHOD TO FIT RAW DATA TO
NORMAL
LOGNORMAL
EXPONENTIAL
GAMMA
WEIBULL
SELECT BEST DENSITY FUNCTION BY KOLMOGOROV SMIRNOV GOODNESS OF FIT TEST
PERFORM ITERATIVE CONVOLUTION SEQUENTIALLY VARYING R_q STANDARDS
C_p
R_q
$(C_p R_q)$
RISK OF ILLNESS
STANDARD (MAXIMUM R_q BEFORE DENYING USE OF BODY OF WATER)
IMPLEMENTATION MANUAL
LITERATURE SURVEY INTERVIEWS SITE VISITS
BSTFIT - DIGITAL COMPUTER PROGRAM
RADOP - DIGITAL COMPUTER PROGRAM
FEDERAL/STATE/LOCAL OFFICIALS TO USE THESE CURVES TO SELECT STANDARDS FOR EACH FACTOR INDEPENDENTLY

Figure 6. Schematic of the Methodology

APPLICATION TO STANDARDS SETTING

The end result is a graph or equation that presents the probability of infection or adverse effect faced by a population entering a water containing various concentrations of the factor of interest. The responsible regulatory agency must then decide on what it considers to be an acceptable risk to the population for that particular factor and set the appropriate standards (the maximum acceptable R_q) to limit that risk.

SECTION VI

VIRUSES AS A FACTOR OF RECREATIONAL WATER QUALITY

During the past 15 years more than 100 new enteric viruses have been discovered, largely through the use of tissue cultures. All of these "new" viruses are excreted in the feces of infected individuals and can be readily demonstrated in urban sewage, particularly in the late summer or early fall (see Table 9). Since these agents are present in sewage, they presumably can find their way into water supplies or surface waters used for recreation and present a hazard of initiating epidemics of water-borne diseases. The relationship between the occurrence of viruses in recreational waters and the real or potential hazard of viral transmission as a consequence of aquatic activity is a multifaceted problem, and one which requires consideration of a variety of interacting factors, some of which are indicated in Figure 7 (after Prier & Riley, 1967).

Several extensive reviews of the general subject of viruses and water transmission have appeared in recent years, notably those of Berg, et al (1967), Chang, (1968), Clarke, et al (1964), and Grabow (1968).

THE VIRUSES OF INTEREST

With the exception of the arboviruses (arthroped-borne), virtually all of the known viruses affecting man cause respiratory tract infections, but only a proportion of them are able to pass through the acid barrier of the stomach and multiply in the intestinal tract. This is the main site of multiplication of the small enteroviruses, adenoviruses, and reoviruses, infection with which may be brought about by the ingestion of virus-contaminated food or water, or by downward spread from the respiratory tract. The prolonged excretion of the virus in the feces following infection causes fecal contamination to be a major factor in their spread (Chang, 1968; Poynter, 1968).

The human enteroviruses are a homogeneous group comprising the polioviruses, the coxsackie-viruses (groups A and B0, and the echoviruses. Most of these viruses produce disease only in a small percentage of the individuals they infect. Low-level, asymptomatic infections are difficult to detect, but may constitue an important source of many enterovirus epidemics not readily ascribable to water transmission (Chang, 1968). When associated with an illness, an enterovirus may be capable of producing any of several clinical entities. This is true also for many of the adenoviruses, some of which are associated with a variety of respiratory diseases (e. g., colds, influenza-like illnesses, bronchitis, etc.), and probably for members of the reovirus (respiratory-enteric-orphan) group as well. The adenoviruses, although usually associated with respiratory ailments, are more readily isolated from the stools than from the throat secretions of infected individuals (Berg, 1964).

The other virus (or, possibly, viruses) of concern is that causing

TABLE 9

Human Enteric Viruses and Diseases
(after Clarke, et al. 1964)

Major subgroup	No. of Types	Disease
Poliovirus	3	Paralytic poliomyelitis, aseptic meningitis
Coxsackie		
Group A	25	Herpangina, aseptic meningitis
Group B	6	Pleurodynia, aseptic meningitis
Infectious Hepatitis	1 (?)	Infectious hepatitis
ECHO	28	Aseptic meningitis, "summer" rash, diarrheal disease
Adenovirus	25	Respiratory and eye infection

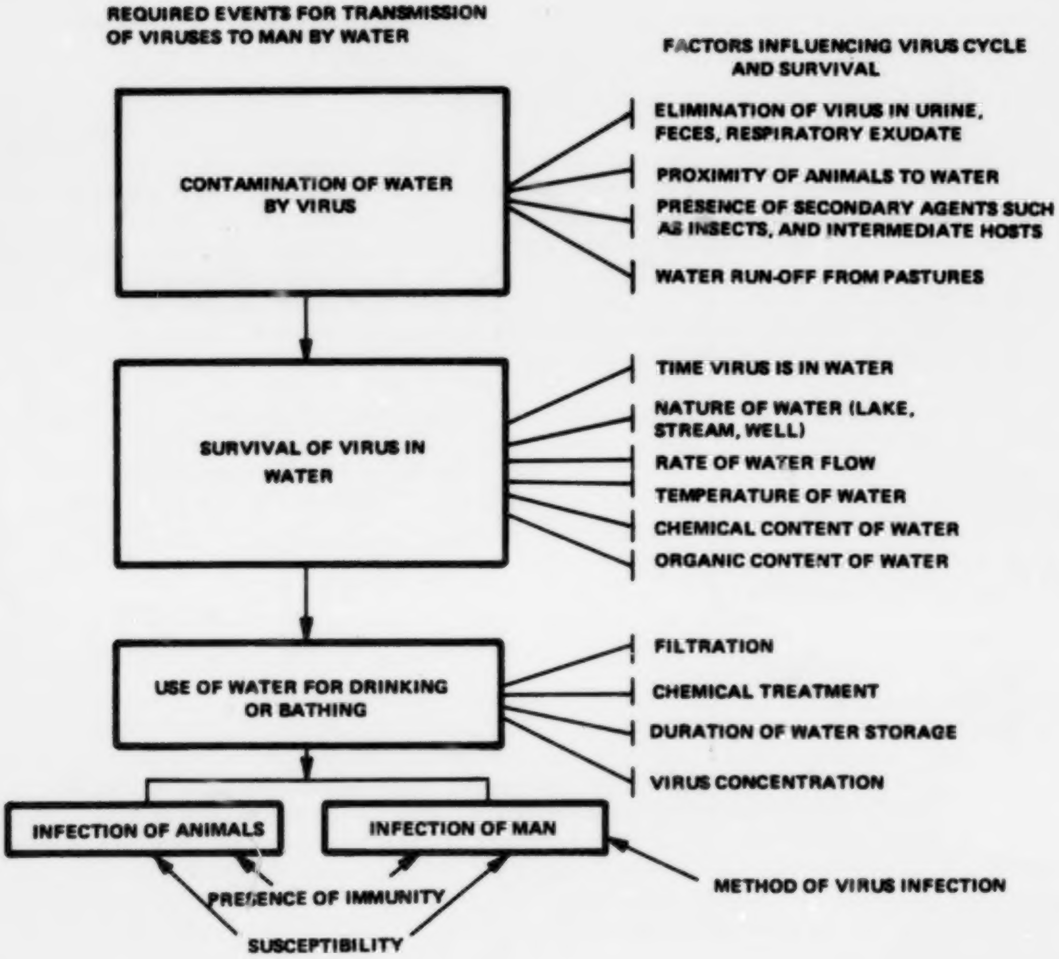

REQUIRED EVENTS FOR TRANSMISSION OF VIRUSES TO MAN BY WATER
FACTORS INFLUENCING VIRUS CYCLE AND SURVIVAL
CONTAMINATION OF WATER BY VIRUS
ELIMINATION OF VIRUS IN URINE, FECES, RESPIRATORY EXUDATE
PROXIMITY OF ANIMALS TO WATER
PRESENCE OF SECONDARY AGENTS SUCH AS INSECTS, AND INTERMEDIATE HOSTS
WATER RUN-OFF FROM PASTURES
SURVIVAL OF VIRUS IN WATER
TIME VIRUS IS IN WATER
NATURE OF WATER (LAKE, STREAM, WELL)
RATE OF WATER FLOW
TEMPERATURE OF WATER
CHEMICAL CONTENT OF WATER
ORGANIC CONTENT OF WATER
USE OF WATER FOR DRINKING OR BATHING
FILTRATION
CHEMICAL TREATMENT
DURATION OF WATER STORAGE
VIRUS CONCENTRATION
INFECTION OF ANIMALS
INFECTION OF MAN
METHOD OF VIRUS INFECTION
PRESENCE OF IMMUNITY
SUSCEPTIBILITY

Figure 7. Factors That May Affect Virus Survival in Water
(After Prier & Riley, 1967)

infectious hepatitis (IH). This is the only known feca ly-excreted virus for which definite epidemiological evidence of water-bor e transmission has been found (Neefe and Stokes, 1945; Mosley, 196). It is also exceptional in that, so far, no tissue cell-culture syste n has been established enabling the IH virus to be isolated in vitro (Ki sling, 1967).

CONTAMINATION OF WATER BY VIRUSES

Gelfand (1961) has summarized data that clearly show the seasonal variation in incidence of enteric viruses in feca samples and sewage. The peak incidence of isolations occurs fro July through October, with August usually yielding the highest nun ber of positive samples. Routine sampling yielded as high as 41.5 p r cent positive sewage samples in one study (Clarke and Kabler, 196). Recovery rates as high as 75 per cent have been reported, how ver, from sewage samples tested during a poliomyelitis epidemic (Wile et al 1962).

Fluctuations in the predominant type of enteric virus etected in sewage frequently occurs (Kelly, et al, 1955, 1956, 960). These changes are said to reflect the epidemic infection in t e community, whether recognized or not, at any given time. Conti ing surveillance of virus in sewage has been suggested as an importan means of monitoring the enterovirus infections of large populations Clarke and Kabler, 1964 and see Table 10).

Viruses occur at very low densities compared to indi ator bacteria, such as the coliform group. It has been calculated (arke, et al, 1964) that in the United States the expected density of virus n sewage would average about 500 virus units per 100 ml, and in poll ed surface water, not more than 1 virus unit per 100 ml. These author determined that the relative enteric virus density to coliform density human feces is about 15 virus units for every million coliforms (a 1 65,000 ratio).

Foliquet and Schwarzbrod (1965) collected 560 sample from various water sources in Meurthe-et-Moselle, France, w th 83 enterovirus strains being recovered as follows:

TABLE 10

Frequency and Types of Enteric Virus*
Isolated from Sewage of the City of Albany

	1957	1958	1959	1960
Poliomyelitis				
Type 1	-	13/36	3/16	3/12
Type 2	1/35	-	-	-
Type 3	11/35	12/36	7/16	5/12
Coxsackie Group B				
Type 3	-	-	-	-
Type 4	8/35	-	-	-
Type 5	10/35	9/36	-	-
ECHO				
Type 1	-	3/36	-	-
Type 3	-	6/36	10/16	1/12
Type 4	7/35	-	-	-
Type 6	-	-	-	1/12
Type 7	3/35	1/36	1/16	-
Type 8	2/35	-	-	-
Type 9	-	3/36	2/16	-
Type 11	2/35	7/36	1/16	-
Type 12	9/35	5/36	-	-
Type 13	-	1/36	-	-
Type 19	-	1/36	-	-

The fractions indicate the number isolated (numerator) from the total number of samples examined (denominator).

*Exclusive of Group A Coxsackie viruses, which are not listed.

Drains: 64 positives of 245 samples (26.1%)
Rivers: 11 positives of 214 samples (5.14%)
Tap Water: 8 positives of 101 samples (7.9%)

The initial concentration of enteroviruses in sewage has been estimated to vary between 20 and 700 Plague-forming units (PFU) per 100 ml, with considerable seasonal variations (Kelly and Sanderson, 1960; Clarke et al, 1962). The concentration of viruses in polluted rivers and streams might be one to two orders of magnitude lower. Thus a monitoring technique for enteroviruses in surface waters should be capable of detecting as few as 1 to 10 PFU/liter.

Paul and Trask (1941) isolated poliovirus from a heavily polluted New England River. These investigators suggested that poliomyelitis occurring down river probably resulted from this water pollution. Toomey et al (1945) recovered polioviruses from an Ohio creek by direct inoculation of water samples into cotton rats.

Rhodes et al (1950) demonstrated that poliovirus persisted in river water for prolonged periods following its addition in a fecal suspension.

Coxsackievirus A5 survived in spring water from extended periods; viral concentration was found to be a function of temperature (Gilcreas and Kelly, 1955).

Clarke et al (1956) and Chang et al (1958) maintained coxsackievirus A2 in water from different rivers under various conditions for long periods.

Metcalf and Stiles (1956) isolated coxsackievirus B4 and echovirus 9 from the eastern oyster in estuary waters as far as 4 miles from the nearest sewage outlet.

At Bathurst, Australia, (Wallace, 1958) clinically typical hepatitis occurred in 6 of 19 students who drank raw water from the Macquarie River. The river was polluted by effluent from the city sewage works located upstream. During the 4 months preceding the outbreak among the students, 10 cases of hepatitis were reported from this community of about 30,000 and all these patients were served by city sewers that emptied into the river. Unfortunately, the quantitative virus content of the water could not be determined, since no laboratory method exists for the propagation of IH virus.

Enteroviruses were isolated from Hudson River water 400 feet down river from the Alaby sewage plant outfall by Kelly and Sanderson (1959).

The problem of isolating and enumerating enterovirus in surface waters is a difficult one, but improved methods now permit the sampling of large volumes of water having low densities of virus contamination (Shuval et al, 1967).

Survival of Virus in Water

Owing to the existence of many unknown factors affecting virus survival in water, the differences in the survival times of different enteroviruses observed in storage are not sufficient to indicate that some enteroviruses survive significantly longer than others. Virus survival in general is longer in treated or "clean" water or in grossly polluted water than in moderately polluted water (Clarke, et al, 1964).

Coxsackie A2 virus survived 61 days in sewage held at 8°C, and 41 days in sewage held at 20°C. The virus survived more than 272 days in distilled water held at 8°C and 41 days at 20°C. However, in the moderately polluted Ohio River water, it survived only 16 days at 8°C and only 6 days at 20°C. Autoclaving the Ohio River water increased the survival time to longer than 171 days at 8°C and more than 102 days at 20°C (Clarke, et al, 1956).

Rhodes, et al, (1950) showed that poliovirus could still be demonstrated in experimentally contaminated river water after 188 days storage at 4°C, but could not be detected after storage at 216 and 315 days.

Using well water known to have been responsible for a water-borne outbreak of infectious hepatitis, Neefe and co-workers (1945) were able to infect human volunteers after the water had been stored for 10 weeks, although there was a considerable prolongation in incubation time with storage time. It appears that only long storage is effective in reducing virus concentrations significantly (Chang, 1968), in the absence of direct measures of viral destruction.

Weber (1958) reported that IH virus remains virulent in ground water at least a month. The virus is quite heat-resistant, especially when suspended in protein. Exposure to 56°C for30 minutes failed to render the virus noninfective, since 3 of 4 men who ingested it contracted the illness (Havens, 1945).

Studies were made by Gilcreas and Kelly (1945) on the relative rates of kill and die-off of coliform bacteria and intestinal viruses by storage in both fresh and salt water, by chlorination, by sewage treatment, and by ultra-violet irradiation. The intestinal viruses used in the tests were Bact. coli B bacteriophage, coxsackie virus, and Theiler virus. Their studies indicated that:

(1) The relative survival of coliform bacteria and viruses in fresh water was almost the same except that viruses survived for much longer periods at low temperature.

(2) In salt water, coliform bacteria died much more rapidly than the viruses.

(3) Chlorination was found to be effective on both coliform bacteria and viruses, but subsequent studies by Kelly and Sanderson (1958) indicated that stronger doses and longer contact periods are required for viruses. In their investigation of five strains of polio and two strains of Coxsackie virus, they found that 0.3 mg/l of free residual chlorine for at least 30 minutes of contact is required for 99.9% inactivation, and that about 10 mg/l of combined residual chlorine for about 60 minutes is required for 99.7% inactivation.

(4) Ultraviolet light was found to be more effective on viruses than on coliform bacteria.

(5) Sewage treatment by primary settling and trickling filters at one plant showed about 90% reduction of coliforms, and about 80% reduction of phage and nearly 100% reduction of Coxsackie viruses.

Clarke and Kabler (1964) determined the relative survival times in raw sewage of 4 enteric viruses and 3 bacterial indicators of pollution (Table 11). The test viruses, with the exception of Coxsackie A9, survived longer than did the 3 test bacteria. All survival times were markedly longer at 4°C than at 28°C.

TABLE 11

Time in Days for 99.9 Per Cent Reduction of Indicated Organism in Raw Sewage

	Temperature	
Organism	28°C	4°C
Poliovirus 1	17	110
ECHO 7	28	130
ECHO 12	20	60
Coxsackie A9	6	12
Aerobacter Aerogenes	10	56
Escherichia coli	12	48
Streptococcus foecalis	14	48

Clarke, et al. (1964), in an extensive study of enteric viruses in water, concluded that the survival times of enteric viruses and indicator bacteria in water depend to a great extent on the nature of the water. Viruses appear to survive longer in water that is relatively unpolluted or grossly polluted. Indicator bacteria (*E. coli*) survival times in surface waters appear to be directly related to the pollution of the water; longest survivals occur in water with the greatest pollution. Metcalf and Stiles (1967) found that survival of viruses in estuary waters is dependent on temperature, pollution levels, and virus identity. In their studies, enteric viruses survived in estuary water for 56 days in winter, and 32 days in the summer.

Waterborne Transmission of Viruses

Opportunities for the spread of waterborne infections may be provided either by recreational bathing facilities or by public or private drinking water supplies. The presence of many bathers in a body of water may provide suitable conditions for the spread of viruses. Outbreaks of sore throats and "pink eye," accompanied by fever and caused by adenovirus-3 in Washington, D. C., in 1954 (Bell, J. A., et al., 1955), and by adenovirus-7 in Toronto, Canada, in 1955 (Ormsby and Aitchison, 1955), were attributed to the use of swimming pools. The spread of vesicular exanthema, caused by Coxsackie A16 virus, was correlated with swimming by Robinson, et al, (1958).

Swimmers at two fresh-water beaches along Lake Michigan showed approximately twice the incidence of gastrointestinal and ear and throat infections as did non-swimmers. A significantly higher incidence of illnesses was reported one week after persons had bathed in water containing 2,300 coliform organisms per 100 ml, in contrast to that observed when the coliform-organism index was 43 (Stevenson, 1953). The risk of contracting infections, however, was not appreciably greater for bathers who swam in salt water contaminated with sewage than it was for those who swam in unpolluted water (Moore, 1960).

When studying water as a possible route of transmission in outbreaks of viral diseases, Mosley (1967) could gather from the literature only 8 episodes of poliomyelitis in which the water route could be presumed, and 50 episodes of infectious hepatitis in which the disease was stated to be waterborne. Only one of the polio outbreaks was considered to be potentially waterborne by Mosley, however. The massive 1955-56 waterborne epidemic of infectious hepatitis in Delhi, India (Viswanathan, 1957), in which more than 28,000 cases were reported, is one of the most often cited of the infectious hepatitis outbreaks. The plant that treated the water at Delhi apparently turned out a safe supply when the water was prechlorinated to a free residual of 0.7 mg/l. When sewage backed up to the waterworks intake, however, sufficient ammonia was present to destroy the free chlorine and to produce a low, combined chlorine residual (Dennis, 1959). The latter treatment nevertheless rendered the water safe according to coliform testing results, but it apparently did not destroy the IH viruses. The data on coliform organisms reported by the new water treatment plant at Delhi would indicate that under unusual circumstances the coliform index may be unreliable for judging the adequacy of modern water treatment processes for destroying or removing the IH virus, and perhaps other enteric viruses (Clarke, et al., 1964). Representative source data for poliovirus and infectious hepatitis outbreaks attributed to contaminated drinking water are contained in the Appendix.

THE MINIMAL INFECTIVE DOSE

Malherbe and Strickland-Cholmley (1967) stated that experimental transmission to human volunteers of the enteric viruses that have been studied usually involves 10^6 $TCID_{50}$ or more. It is generally recognized, however, that infection regularly follows the oral administration of 10^5 $TCID_{50}$ of attenuated poliovirus strains, and there is some evidence that as little as 10 $TCID_{50}$ of attenuated poliovirus may cause infection.

Plotkin and Katz (1967) reviewed the information available concerning the relationship between infectivity for tissue cultures and infectivity for man and animals of viruses administered by the oral, respiratory, or conjuncival routes. These authors contend that one infective tissue culture dose is sufficient to infect man if it is placed in contact with susceptible cells. The relationship between one virus and one cell is complicated in the intact host, however, by a variety of host defense factors, which accounts for the variations observed in the amounts of virus actually required to produce discernible infection in man (Beard, 1967).

Since extracellular virus cannot reproduce itself, it seems unlikely that the amount of virus of human origin in water sources can ever be large, except at times of major epidemics, but if small amounts are capable of infecting, then their presence in food or water, however dilute, assumes importance (Poynter, 1968).

The available data is consistent with the view that the human subject is not less susceptible than the tissue-culture. Koprowski (Table 12) has shown that 2 PFU's of attenuated poliovirus 1 produced infection in 2 out of 3 subjects, while Plotkin and Katz (Table 13) infected two out of three infants with 10 TCD_{50} of attenuated poliovirus 3, and obtained evidence that even 1 TCD_{50} of this strain may be infective for infants.

TABLE 12

Infection of Human Volunteers with Attenuated Poliovirus 1

(after Koprowski, 1955; 1956)

Dose (PFU)	No. Infected/ No. Fed	Per cent Infected
0.2	0/2	0.0
2.0	2/3	66.7
20.0	4/4	100.0
200.0	4/4	100.0

TABLE 13

Infectivity of Enteroviruses for Man by the Oral Route

(after Plotkin & Katz, 1967)

Virus	Dose	Result	Reference
Poliovirus 1 (SM)	2 PFU	2/3 infected	Koprowski (1955, 1956)
Poliovirus 1 (SM)	20 PFU	1/2 infected	Koprowski et al (1956)
Poliovirus 2 (P712)	100 TCD_{50}	Infection (no details)	Sabin (1956)
Poliovirus 3 (Fox 13)	10 TCD_{50}	2/3 infected	Plotkin & Katz (1967)

Sabin found 10^6 TCD_{50} of poliovirus per gram of feces in human stools. Neefe, et al (1945) estimated that there were 10^4 to 10^5 infectious doses of IH viruses per gram of feces from human cases. Other estimations of viral content in feces have been of the same order of magnitude or less.

Exceedingly minute amounts of infective excreta can cause infectious hepatitis when taken orally. Human volunteers have developed it from as little as 10^{-20} dilution of infected feces (Stinger, 1955). According to Clarke and Chang (1959), a 30-minute chlorine contact period protected all of 12 volunteers, when the IH virus was suspended in distilled water at room temperature, pH range 6.7-6.8, and with initial and final free chlorine residuals of 3.25 and 0.4 mg/l.

DEVELOPMENT OF CRITERIA

Virus Factor to Criteria Conversion

A considerable amount of information has been reviewed dealing with experimental virus infections using human volunteers. Although many of the reported studies were carried out for purposes other than to determine infective dose (most were centered around vaccine development), there were sufficient base line experiments to provide inputs for developing a dose effect curve.

Various ways of measuring the inoculum dose given to volunteers were reported. These included particle counts, infective serum dilutions, plaque forming units, tissue culture units, and human infective doses. The $TCID_{50}$ dose was most frequently reported or in some cases the data reported could be used to calculate this dosage. The $TCID_{50}$ represents the tissue culture infective dose that effects 50 percent of the roll tubes of the specified tissue cells when the usual 10 fold dilution series is carried out. Effects may be reported as cytopathic effects (CPE), or, more usually, as the results of the hemadsorption test.

If one assumes that the cultured tissue cell responds in much the same way as normal cells do in vivo (and there is no reason to assume otherwise) to an infection by a given virus, certain simplifications are possible. By utilizing $TCID_{50}$ to measure inoculum dosage, the data are normalized to a common base irrespective of differences in infectivity of the various viruses. For example, 10^5 particles may be required to infect 50 percent of the cultures with one strain and 10^3 with another. By reporting both sets as 1 $TCID_{50}$ dose however, this difference is canceled out and the viruses can be grouped together for statistical purposes. This was done in the present study. Data on volunteers, where the inoculum dose was reported in units of $TCID_{50}$, were all grouped together in order to prepare a dose effect curve for virus exposures, egg infective doses were assumed equal to tissue culture infective doses. In this manner a table was prepared listing 812 volunteer exposures ranging from 2×10^6 to 1×10^{-1} $TCID_{50}$ does (Table 14).

TABLE 14

Viral Dose-Response of Humans

Virus	Dose, $TCID_{50}$	Number Infected	Route	Investigator
Parainfluenza 2	2.0×10^6	6/6	Nasal	Taylor-Robinson
Rhinovirus	6.3×10^5	2/2		Cate
	3.2×10^5	4/5		Taylor-Robinson
Parainfluenza 2	2.0×10^5	9/9		Taylor-Robinson
Rhinovirus	1.6×10^5	2/3		Cate
Adenovirus	1.5×10^5	2/2	Nasal	Hitchcock
Parainfluenza 3	1.5×10^5	3/4	Nasal	Tyrrel
Hemadsorption 3	1.0×10^5	12/17		Kapikian
Influenza	8.0×10^4	10/10	Nasal	Knight
Equine influenza	7.9×10^4	4/5	Nasal	Knight
Rhinovirus	6.3×10^4	4/5		Cate
Inclusion conjunctivitis	6.0×10^4	2/2		Jawetz
Inclusion blennorhea	4.0×10^4	1/1		Jones
Parainfluenza 2	2.0×10^4	5/6		Taylor-Robinson
Parainfluenza 3	1.5×10^4	2/4		Tyrrel
Adenovirus	1.5×10^4	2/3	Nasal	Hitchcock
JH virus	4×10^3 - 1.4×10^4	23/69		Jackson
2060 virus	4×10^3 - 1.4×10^4	25/90		Jackson
Influenza	1.0×10^4	5/5	Nasal	Knight

TABLE 14 (continued)

Virus	Dose, $TCID_{50}$	Number Infected	Route	Investigator
Inclusion conjunctivitis	6.0×10^3	3/3		Jawetz
Rhinovirus	3.2×10^3	4/4		Cate
Adenovirus	2.5×10^3	2/2	Nasal	Hitchcock
Parainfluenza 2	2.0×10^3	0/3		Taylor-Robinson
Influenza	1.9×10^3	3/3	Nasal	Knight
Influenza	1.6×10^3	0/3	Nasal	Knight
Adenovirus	1.5×10^3	1/2	Nasal	Hitchcock
Parainfluenza 3	1.2×10^3	3/6		Tyrrel
Coxsackie A_{21}	1.0×10^3	2/3		Knight
Coxsackie A_{21}	832	1/1		Tyrrel
Influenza	790	7/7	Nasal	Knight
Coxsackie A_{21}	676	5/6	Inhalation	Couch
Rhinovirus	630	17/32		Cate
Inclusion conjunctivitis	600	2/2		Jawetz
Coxsackie A_{21}	500	8/9		Knight
Adenovirus	250	0/2		Hitchcock
Parainfluenza 2	200	0/2		Taylor-Robinson
Coxsackie A_{21}	160	2/2		Knight
Influenza	158	0/1	Nasal	Knight
Parainfluenza 1	150	3/5		Tyrrel

TABLE 14 (continued)

Virus	Dose, $TCID_{50}$	Number Infected	Route	Investigator
Adenovirus	150	1/4		Hitchcock
Inclusion conjunctivitis	125	4/4		Jawetz
Poliovirus 3	100	4/4		Plotkin & Katz
Poliovirus	30-100	7/9		Cate
Coxsackie A_{21}	83	2/2		Reichelderfer
Hemadsorption 2	80	25/32		Reichelderfer
Coxsackie A_{21}	71	4/4	Inhaled	Couch
Rhinovirus	66	1/1	Inhaled	Cate
Rhinovirus	60	1/1	Inhaled	Cate
Inclusion conjunctivitis	60	5/6		Jawetz
Coxsackie A_{21}	54	2/2	Inhaled	Couch
Coxsackie A_{21}	50	2/2		Knight
Coxsackie A_{21}	49	2/3	Inhaled	Couch
Coxsackie A_{21}	47	3/4	Inhaled	Couch
Coxsackie A_{21}	28	2/3	Inhaled	Couch
Rhinovirus	20	1/1	Inhaled	Cate
Measles	20	28/31		Okuno
Coxsackie A_{21}	18	2/4	Inhaled	Couch
Coxsackie A_{21}	16	1/3	Inhaled	Couch
Rhinovirus	16	5/5	Inhaled	Cate

TABLE 14 (continued)

Virus	Dose, $TCID_{50}$	Number Infected	Route	Investigator
Adenovirus	15	0/2	Nasal	Hitchcock
Parainfluenza 1 (HA 1)	15	4/11		Tyrrel
Parainfluenza 3	15	0/6		Tyrrel
Inclusion conjunctivitis	12.5	2/3		Jawetz
Poliovirus 3	10	2/3		Plotkin & Katz
Measles	10	18/21	Inhalation	Plotkin & Katz
Measles	10	21/43		Okuno
Measles	6.0	18/38		Okuno
Measles	6.0	18/26		Plotkin & Katz
Coxsackie A_{21}	6.0	2/6		Jawetz
Inclusion conjunctivitis	6.0	0/3		Jawetz
Measles	2.0	14/29		Plotkin & Katz
Parainfluenza 1 (HA 1)	1.5	2/2		Tyrrel
Inclusion conjunctivitis	1.25	1/3		Jawetz
Measles	1.0	8/35		Plotkin & Katz
Measles	1.0	1/8		Okuno
Measles	0.6	15/27		Okuno
Measles	0.6	1/8		Plotkin & Katz
Inclusion conjunctivitis	0.6	0/3		Jawetz
Measles	0.2	1/13		Okuno

TABLE 14 (continued)

Virus	Dose, $TCID_{50}$	Number Infected	Route	Investigator
Measles	0.2	1/7		Plotkin & Katz
Measles	0.1	0/21		Plotkin & Katz
Measles	0.1	0/23		Okuno

Development of the Dose Response Curve

The data from the infectivity studies with 812 volunteers resulted in 413 data points which were used to construct a dose esponse curve for viruses. When all 413 data points were used in the rogram, the data scatter was such that a fit was not possible.

The fact that the dosages measured by tissue culturtitration are estimates of the dose fed rather than exact measure a histogram was constructed to order this data. From the histogram 25 points relating dose fed to number of individuals infected were randmly selected (Figure 8). This represented the test sample that ws to be used to develop the capabilities vector (C_p) or the ability of e population to withstand a given challenge dose of virus.

In line with the methodology previously discussed, t probability distribution of the sampled population was estimated The collected information was analyzed using the BSTFIT compute program and the Kolmogorov-Smirnov test indicated that the lognorml distribution was the most appropriate (Table 15).

It is conceded that this method of sampling from a histogram does not take full advantage of the potential of the availabl data. It is possible that the data could be grouped according to fferent regions of the world reflecting varying susceptibility to differe viruses in population groups. There may also be a varying regional sceptibility due to the effectiveness of recent immunization programs. An attempt should be made to see if such variances appear in the data. It may also be argued that a population is in general susceptible to e viruses that appear in its waters or else how could the viruses bemaintained without infections occuring? Thus it can be proposed tha only the viruses to which the population is susceptible occur in the ar's waters. The use of the TCD_{50} as the measure of dosage would compensate for regional differences by implying that each population respols to its indiginous viruses and that infective doses measured as TCD_{50} ill not vary from population to population.

Based on this best-fit analysis, the data were plotted s the lognormal distribution using the 50% tissue culture infective dos ($TCID_{50}$) vs the percent cumulative probability of infection, alongwith the 95% upper and lower confidence limits. This represents e C_p vector, as presented in Figure 8.

Development of Virus Distribution Vector for a Given Water Body

The virus requirements or distribution to be expected in a given polluted water were developed from a study of a stream carril out by S. Grinstein, J. Melnick, and C. Wallis, (1970). The stream (Bras Bayou) passes through a Houston, Texas, residential area and a 10 ile stretch, receiving various treatment plant effluents, was selected fo study. A map is

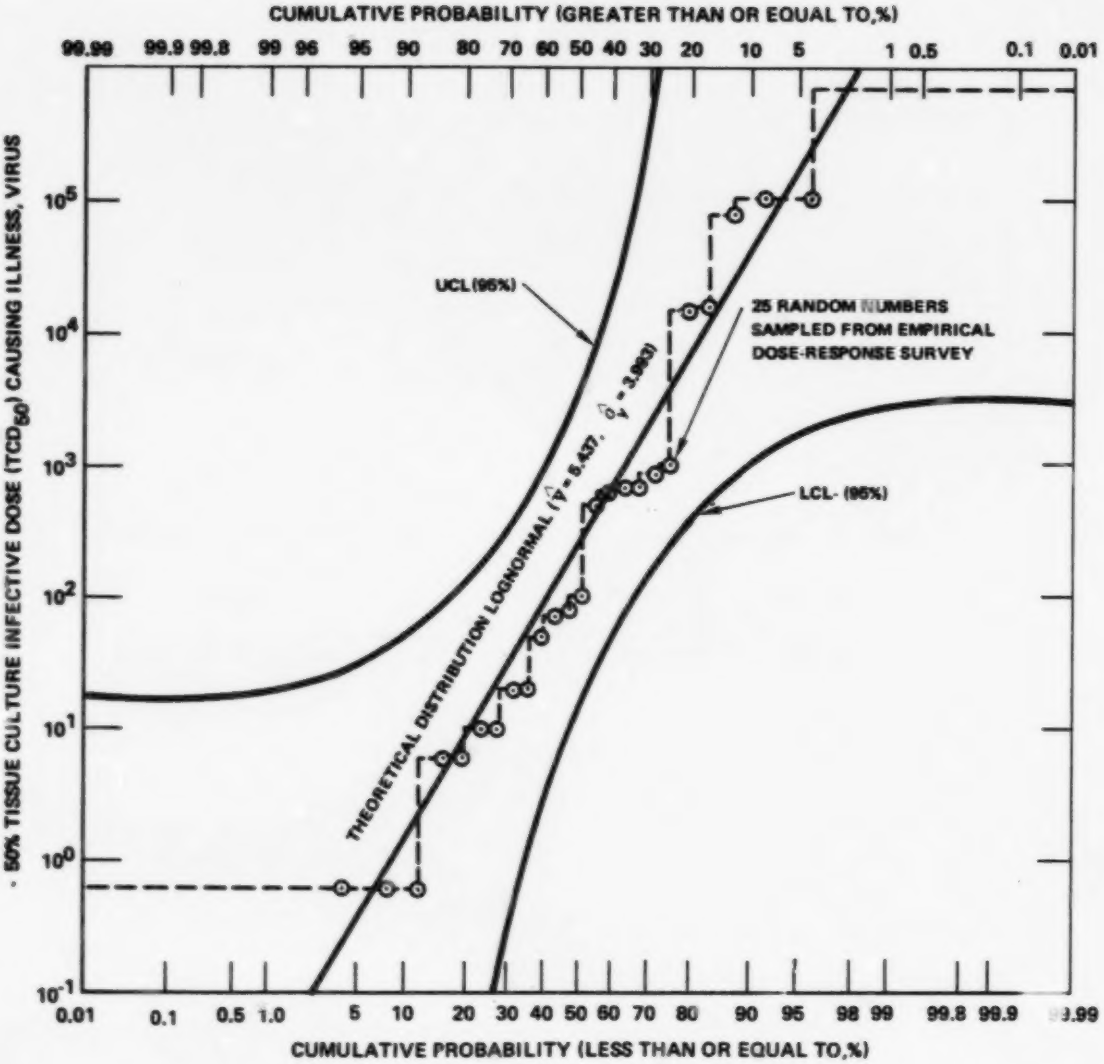


Figure 8. Virus Capabilities Vector (C_p)

TABLE 15

Virus Capability Vector (C_p)
Using 25 Random Samples

NAME OF DISTRIBUTION	"d" STATISTIC	PROBABILITY DATA CAME FROM CITED DISTRIBUTION
Normal	0.411	0.000
Lognormal	0.116	0.888
Exponential	0.735	0.000
Gamma	0.234	0.129

presented in the original article. The data from collection points 10 and 11 were omitted because the stream broadens into a river at that point. The samples from the sewage treatment plants were also omitted because the samples were taken at the beginning of the treatment process and were not representative of effluent discharge. In all, 39 data points were used to develop the probability distribution of viruses along the stream's course.

As in the previous analysis, a histogram of the data was prepared, this time relating number of viral PFU per gallon to the frequency of isolations of the various viral concentrations. Twenty-five points were selected at random from the histogram and a BSTFIT analysis carried out. For this particular data, the Kolmogorov-Smirnov "d" test determined that a 3-parameter Weibull was the most appropriate representation of the sampled universe (Table 16). This 3-parameter Weibull is a recent advancement in the computer program (replacing a less exact, 2-parameter fit) and provides the potential for more accurate fitting of data.

The 3-parameter Weibull virus distribution is presented in Figure 9 along with the upper and lower 95% confidence limits and the cumulative histogram of the 39 data points. This plot indicates that 80 or more PFU/gal. were present 50% of the time. An estimate of the virus risk in a given water can be made by carrying out a convolution.

The Convolution and Development of Risk versus Concentration Criteria

The two elements, host susceptibility (C_p) and factor concentration distribution (R_q), essential as inputs into the methodology for assessing risk, are now available. The two probability functions are operated upon within the computer program and the probability density function of the difference, ($C_p - R_q$), determined. The negative area of this convolution (to the left of the zero point-see Figure 4) thus represents the risk to the recreationist. At the same time, the requirements vector (concentration distribution in the water) is truncated at points from 1 through 7 standard deviations (see Figure 5) from the mean to provide an evaluation of how much the risk is decreased as the entry to the water is limited whenever these levels are exceeded. The results of this analysis are presented in Table 17. The term "basic" for the C_p vector implies that the dose response distribution was used as originally derived in the analysis for each truncation of R_q.

In carrying out the convolution it was assumed that a recreationist would imbibe 10 ml of water in the time he is exposed (Streeter, 1951). From the analysis, the polynomial equation describing the risk versus concentration relationship was derived. The analysis of variance for the various polynomial fits selected the quadratic regression as the most applicable.

TABLE 16

Virus Requirement Vector (R_q) Formulated from Melnick Data (Measurement Stations 1, 2, 4, 6, 7, 9)

NAME OF DISTRIBUTION	"d" STATISTIC	PROBABILITY DATA CAME FROM CITED DISTRIBUTION
Normal	0.1632	0.250
Lognormal	0.1012	0.819
Exponential	0.1038	0.794
Gamma	0.0915	0.900
Weibull	0.0764	0.977

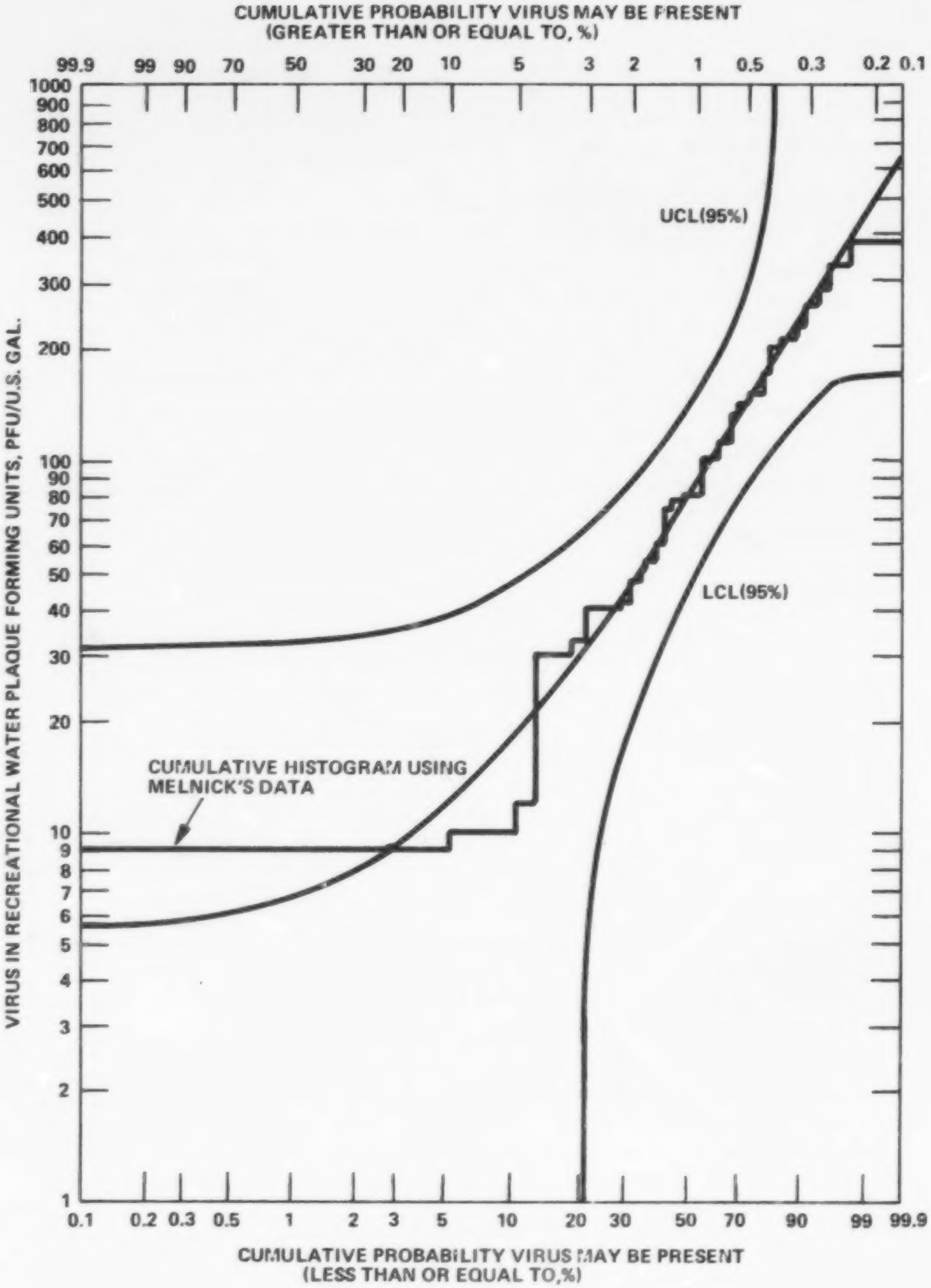


Figure 9. Virus Requirements Vector (R_q)

This equation, presented in Table 18, was then used to prepare the graph in Figure 10 displaying the risk to population using Brays Bayou as a function of the virus concentration. The very narrow 95% upper and lower confidence limits indicate that a true physical relationship exists and that the mathematical model is a good representation of this relationship.

It will be noted that the virus data is in the form of PFU's, while the original dose response curve was reported in terms of $TCID_{50}$. The $TCID_{50}$ units were converted over to PFU's according to the method of Davis, et al (1968), where PFU = 0.7 TCD_{50}. This conversion was carried out to put the dose effect data into the same units as reported for the water concentration distribution. The PFU-assay is finding favor with pollution control authorities, who consider that it is a direct count of infectious units and thus comparable to a bacterial plate count.

Examination of the information presented shows that a linear extension of the dose-response curve predicts a 0.01% (1/10,000) probability of infection if 4.7×10^{-5} PFU (1×10^{-4} TCD_{50}) are ingested. For a recreationist, this dosage would have to be contained in the 10 ml he imbibes.

Using the distribution pattern evident in Brays Bayou in Houston as an example, the convolution of the dose response data and of the varying virus concentrations has been presented. This reveals that in Brays Bayou the 0.01% risk level is at 6.5 PFU/l (Figure 11). In other words, whenever 6.5 PFU/l are detected at any of the sampling points on Brays Bayou a disease incidence of 1 case out of 10,000 recreationists can be expected.

This analysis is further extended by utilizing the coliform-virus relationship for polluted waters presented by Clarke et al (1964) and Sanderson and Kelly (1961). Although the information is based on a limited amount of sampling and a probability distribution has not been carried out, this data is the best available at the present time. These workers estimate a ratio of approximately 50,000 coliform bacteria per PFU. This virus-coliform ratio is plotted below the virus concentration in Figure 11 and reveals that for a 1/10,000 risk level of disease, 35,000 MPN coliform/100 ml can be tolerated in Brays Bayou.

The risk based on fecal coliforms can be similarly estimated if the fecal coliform-total coliform relationships developed by Strobel (1968) for Shellfish waters in New York is assumed to hold true in Brays Bayou. By plotting this relationship in Figure 11, a fecal coliform count of about 18,000 MPN/100 ml presents a 1/10,000 risk of illness due to virus.

This analysis is presented as an orientation to existing standards. At present the maximum amount of coliform organisms permitted by any state for recreational waters is 5000 MPN coliform/100 ml. However it must be emphasized that for determining the actual risk involved in Brays Bayou, the virus-coliform and coliform-fecal coliform relationships will actually have to be determined for that particular water. Once this is done, a chart similar to Figure 11 can be constructed for that water.

TABLE 17

Limitation of Virus Exposure and the Resultant Risk to a Population

CONVOLUTION $C_p - R_q$		CRITERIA (Truncation Point of R_q Vector)	P (Illness)
Virus (PFU)	Virus (PFU)	Virus (PFU/l)	%
Basic	$\bar{x} + 7\ \sigma$	216	0.193
	$\bar{x} + 6\ \sigma$	189	0.191
	$\bar{x} + 5\ \sigma$	163	0.183
	$\bar{x} + 4\ \sigma$	136	0.168
	$\bar{x} + 3\text{-}1/2\ \sigma$	123	0.158
	$\bar{x} + 3\ \sigma$	109	0.147
	$\bar{x} + 2\text{-}1/2\ \sigma$	96	0.134
	$\bar{x} + 2\ \sigma$	83	0.120
	$\bar{x} + 1\text{-}1/2\ \sigma$	69	0.104
	$\bar{x} + 1\ \sigma$	56	0.087
	$\bar{x} + 1/2\ \sigma$	43	0.068
Basic	$\bar{x}$	29	0.048

With this information it is now possible to determine the distribution of viruses in any given water and, using the dose-response probability distribution, construct a risk curve for that water.

TABLE 18

Derivation of Equation to Describe Recreationist Risk as a Function of Virus Criteria

Equation of Polynomial Best Fit	Analysis of Variance		
	F* Calculated	F Minimum allowable from table**	
		Level of significance	
		5%	1%
$Y = -2.7453 \cdot 10^{-3} + 1.84965 \cdot 10^{-1} X - 4.3744 \cdot 10^{-2} X^2$	13,171	5.12	10.56

---

[*] F distribution, which is the distribution of the ratio of two variances.

[**] From tables of critical values of F at 5% and 1% levels. When the calculated value for F exceeds the critical value, the answer is significant.

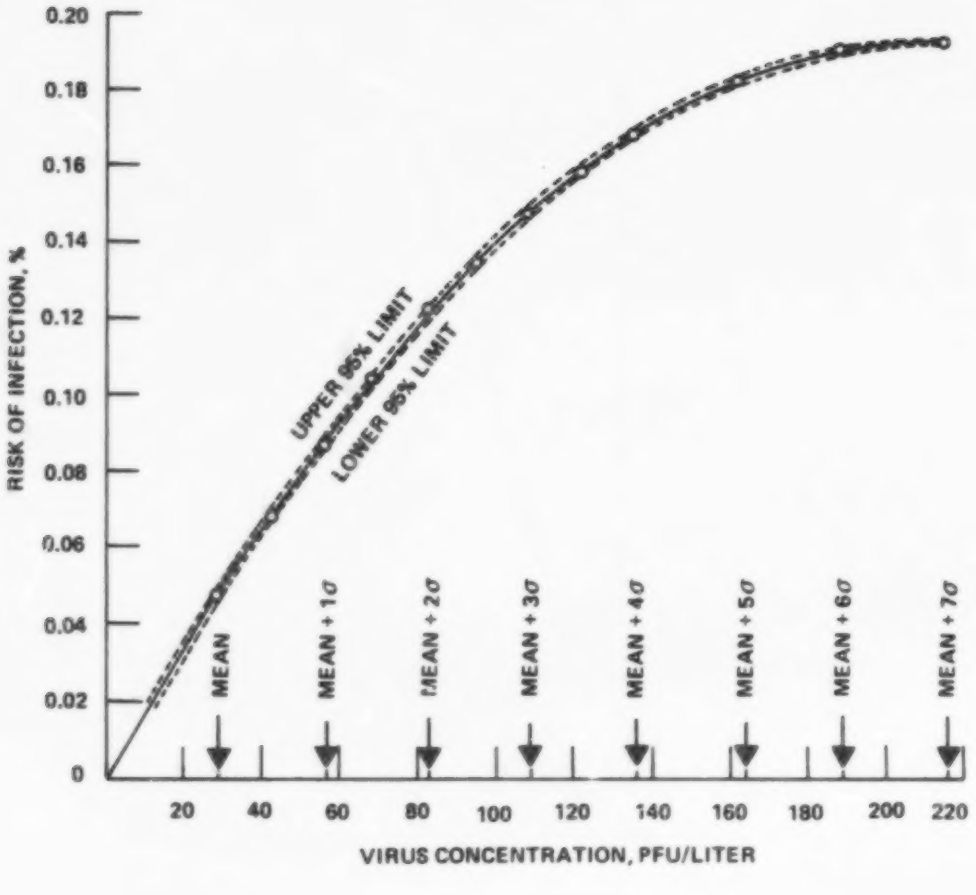


Figure 10. Criteria for the Presence of Virus in a Recreational Water

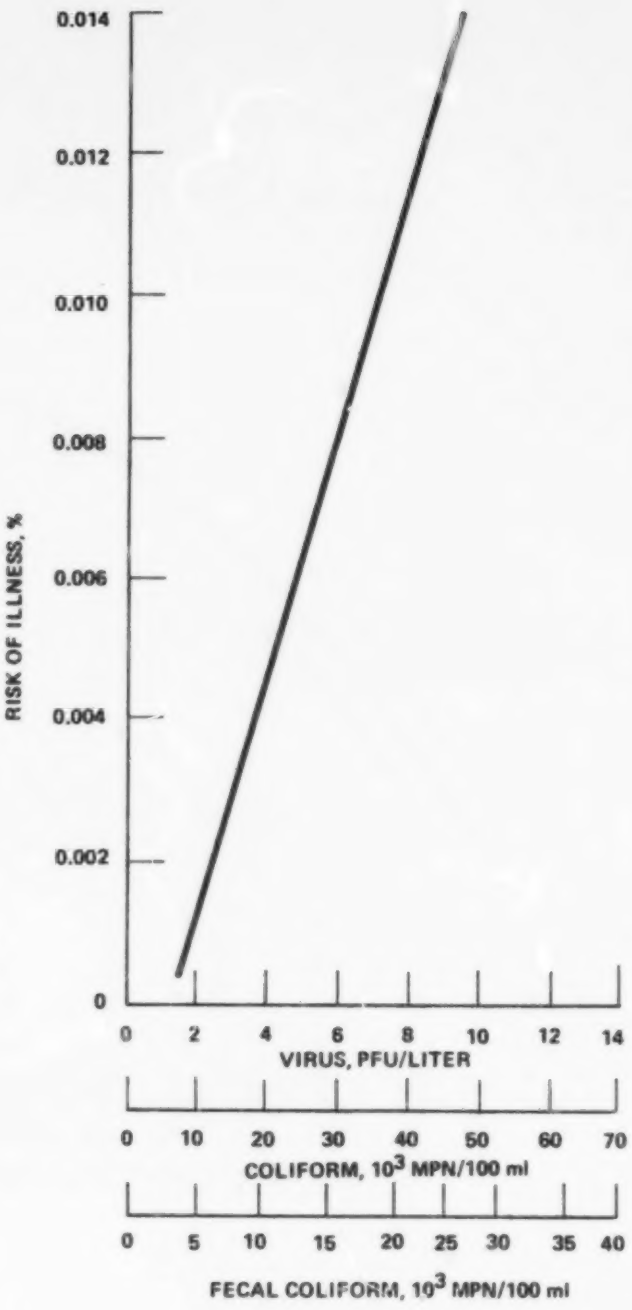


Figure 11. Coliforms and Fecal Coliforms as Indicators of Virus Risk

SECTION VII

SALMONELLA AS A FACTOR OF RECREATIONAL WATER QUALITY

DISEASE AND ETIOLOGICAL AGENTS

Salmonella infections result from the direct or indirect transmission by a suitable vehicle of an infecting dose of species of the genus *Salmonella* from an existing reservoir of infection to a susceptible individual. These infections are of two types, namely, salmonellosis proper and the enteric fevers.

Salmonellosis is characterized by a sudden, usually transient, stormy gastrointestinal disturbance, following a short incubation period. The enteric fevers, which include typhoid and paratyphoid fevers, are evidenced by a gastroenteritis similar to that produced by other salmonella species, as well as a generalized infection of the small intestine, involving invasion of the blood stream, with all of the symptoms occasioned by septicemia. The incubation period is usually long (8-15 days).

Figure 12 is a diagrammatic representation of some of the parameters to be considered in the case of *Salmonella* as a factor in recreational water quality.

SOURCES OF INFECTION

Salmonellae fall into three groups with respect to their distribution and their relationship to human disease:

(1) The first group contains those that are primarily human pathogens and includes *S. typhosa*, (synonyms: *Bacterium typhosum*, *Bacillis typhosus*, *Bacillus typhi abdominalis*; *Bacterium typhi*, *Eberthella typhi*, and *Salmonella typhi*), *S. paratyphi*, *S. schottmuelleri*, and *S. hirschfeldii*. Of these *S. typosa* is the most important, because of the severity of the disease it produces. *S. schottmuelleri* is the most common in the United States, *S. paratyphi* is occasionally isolated, and *S. hirschfeldii* is very rare.

(2) The second group is made up of organisms that are primarily pathogenic for animals, including birds, but which may occasionally cause disease in man. It contains the majority of the *Salmonellae*. The relative incidence of these species in human infections varies in different geographical areas, anddepends in large part on the number of person involved in the particular outbreaks studied.

The U.S. Public Health Service maintains routine surveillance of isolations of *Salmonellae* reported by the various state health agencies. During the period January to June, 1969, the total number of *Salmonella* isolations reported from humans was 8,645. The ten most frequently isolated serotypes are listed in Table 19, with total numbers reported for the period.

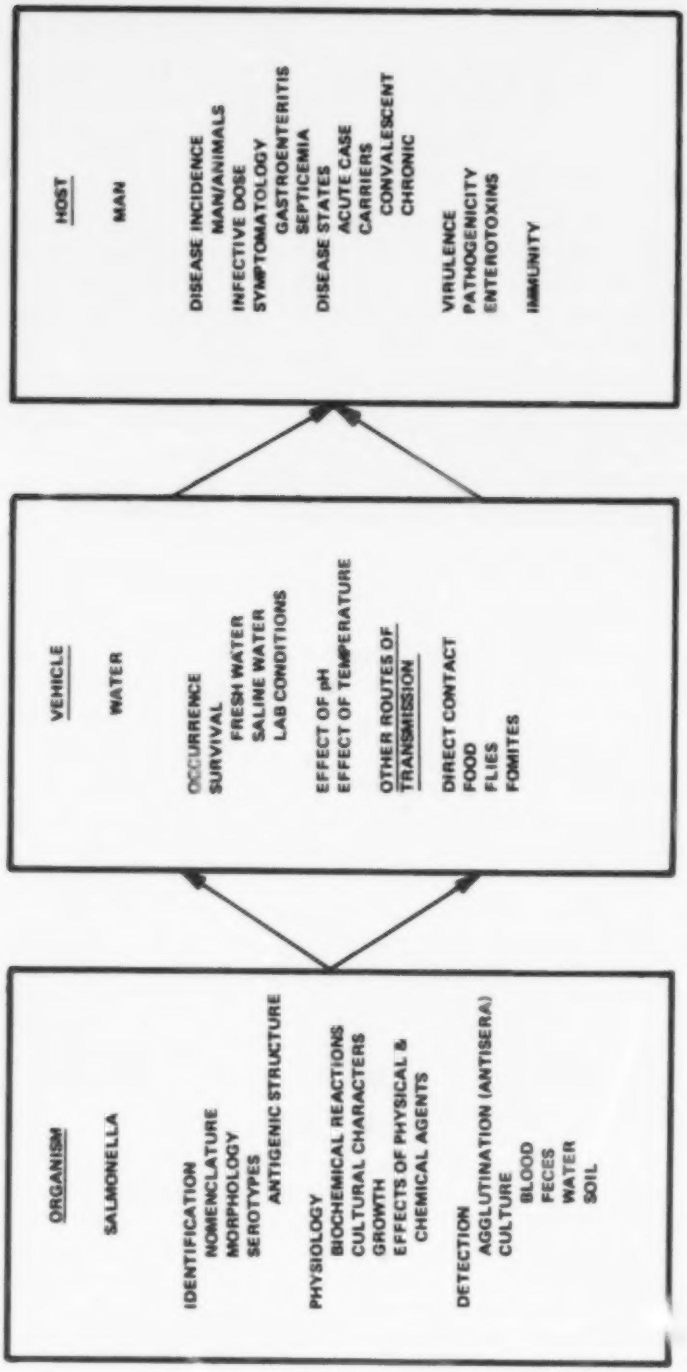

ORGANISM
SALMONELLA
IDENTIFICATION
NOMENCLATURE
MORPHOLOGY
SEROTYPES
ANTIGENIC STRUCTURE
PHYSIOLOGY
BIOCHEMICAL REACTIONS
CULTURAL CHARACTERS
GROWTH
EFFECTS OF PHYSICAL &
CHEMICAL AGENTS
DETECTION
AGGLUTINATION (ANTISERA)
CULTURE
BLOOD
FECES
WATER
SOIL
VEHICLE
WATER
OCCURRENCE
SURVIVAL
FRESH WATER
SALINE WATER
LAB CONDITIONS
EFFECT OF pH
EFFECT OF TEMPERATURE
OTHER ROUTES OF
TRANSMISSION
DIRECT CONTACT
FOOD
FLIES
FOMITES
HOST
MAN
DISEASE INCIDENCE
MAN/ANIMALS
INFECTIVE DOSE
SYMPTOMATOLOGY
GASTROENTERITIS
SEPTICEMIA
DISEASE STATES
ACUTE CASE
CARRIERS
CONVALESCENT
CHRONIC
VIRULENCE
PATHOGENICITY
ENTEROTOXINS
IMMUNITY

Figure 12. Some Epidemiological Parameters of Salmonellosis in Man

TABLE 19

Ten Most Frequently Reported *Salmonella* Serotypes from Humans, January to June, 1969

typhimurium	2,453
enteriditis	819
infantis	559
heidelberg	556
newport	546
thompson	489
saint Paul	383
typhi	227
blockley	215
derby	142

(Source: U.S. Public Health Service Morbidity & Mortality Report, 18:224, 329).

(3) In the third group are found those Salmonellae that are known to be pathogenic only for animals or birds. This group has rapidly diminished as more and more of these species have been found to cause disease in man. S. gallinarum, S. abortivoequina, and S. abortusovis are the important organisms in this group.

That Salmonellae are widely dispersed and occur frequently among the various species of lower animals is evident from the numerous reports in the literature. This statement applies not only to the host-adapted types (the so-called "primary salmonellosis" of many species) but to the nonhost-adapted types as well. Not only do some types occur both in animals and man, but there is a distinct correlation between the presence of the organisms in lower animals and in the human population in any given locality, although the methods whereby the organisms are transmitted from animals to man and vice versa may vary from one region to another depending on environmental sanitation and habits of the population (Edwards, 1956; 1958).

Thus, in order to appreciate fully the ramifications of the epidemiology of infections due to nonhost-adapted types of Salmonellae, it is necessary to consider animal reservoirs of infection; excretion of the bacteria by human cases, convalescents, and carriers; the occurrence and survival of the organism in the environment; and the methods whereby they are transmitted among and between man and the lower animals.

The Salmonelloses and enteric fevers (including typhoid) are outstanding examples of diseases that may be transmitted by individuals who, to all appearances, are normal and healthy, but are dangerous because they harbor infectious agents. In the case of typhoid fever, many epidemics have been traced to persons who excrete typhoid bacilli in their feces, although they show no outward manifestations of the disease. About one-third of the acute cases discharge typhoid bacilli for 3 weeks after onset, and about 10% for 8-10 weeks. The latter group are classed as convalescent carriers. Some typhoid cases may continue to excrete the organisms for several months, years, or their lifetime, to become chronic asymptomatic carriers. Similar conditions exist in the case of the salmonelloses proper, as evidenced by the frequency with which asymptomatic persons are revealed to excrete the organisms in their feces. The experiments of McCullough and Eisele (1951, series of 4 papers) demonstrated that symptomless Salmonella infections could be induced in human volunteers, as evidenced both by fecal excretion and rise in agglutinin titer.

The significance of the carrier state to our studies lies in the fact that the occurrence of Salmonella in recreational waters that have been polluted by human or animal fecal matters is not always directly related to frank (symptomatic) disease in the human or animal populations contributing that factor, and that epidemiological studies that do not take into account the symptomless carriers may fail to reveal the source of infection (endogenous or exogenous) derived from aquatic activity. The carrier, however, continuously excretes very large numbers of pathogens in his feces and thus poses a threat to the population at large (Table 20).

TABLE 20

Number of *S. typhosa* and *S. paratyphi* B
in a Gram of Feces of Carriers

Typhoid Carriers			
	Known Duration of Carrier State	Number of organisms/g feces	
Carrier	(Years)	*S. typhosa*	Bact. coli, etc.
1	3	4,500,000	7,000,000,000
2	3	4,000,000	7,000,000,000
3	3	550,000	1,400,000,000
4	12	45,000,000	5,000,000
5	12	2,500,000	20,000,000
6	12	1,000,000	100,000,000
7	12	600,000	700,000,000
8	12	500,000	300,000,000
9	12	500	300,000,000
10	12	500	450,000,000
11	12	500	120,000,000
12	12	500	650,000,000

Paratyphoid Carriers			
	Known Duration of Carrier State	Number of organisms/g feces	
Carrier	(Years)	*S. paratyphi* B.	Bact. coli, etc.
13	1	12,000,000,000	1,550,000,000
14	1	2,000,000,000	150,000,000
15	1	500,000,000	10,000,000
16	1	300,000,000	8,500,000
17	1	200,000,000	2,450,000
18	1	90,000,000	40,000,000
19	1	20,000,000	300,000,000
20	1	100,000	10,000,000
21	12	25,000,000	2,500,000,000
22	18	50,000,000	250,000,000
23	18	10,000,000	20,000,000
24	20	500,000	110,000,000

MODES OF TRANSMISSION

Outbreaks of typhoid fever have been traced most often to drinking water and milk supplies contaminated with *S. typhosa*. Other means of transmission are flies, infected shellfish, infected food, fomites, and carriers, although *S. typhosa* is sharply distinguished from all other *Salmonellae* by its long-recognized association with water. As human excremental organisms do not multiply in water but merely survive in ever-diminishing numbers (see below) for periods varying with the condition of the water, this association of *S. typhosa* infection with water is another indication that the infecting dose of this serotype is smaller than that of other serotypes (McCoy, 1964).

In 1961, an outbreak of typhoid fever was reported from Australia (Anon (typhoid), 1961) that was traceable to swimming at a contaminated beach. A nearby sewage plant effluent was implicated as the source of infection in a group of ten persons. In a five-year study of sea bathing water and illness of bathers, the British Committee on Bathing Beach Contamination encountered small numbers of *Salmonella* in the high proportion of samples. Although the most frequently isolated *Salmonella* was *S. paratyphi* B, a total of 33 different species was encountered (Moore, 1959; Khait, 1960).

In Argentina, Palazzolo, et al (1954) isolated *S. newport*, *S. typhimurium*, *S. muenster*, *S. bredeney*, *S. montevideo*, and *S. anatum* from children suffering from enteric disorders. In a canal epidemiologically related to the homes of the children, they recovered 11 serotypes of *Salmonella* in 83% of the samples taken, six of which were identical with those recovered from the children. Similarly, Steinigh (1953) recovered *S. panama* from a harbor water in the neighborhood of a patient infected with the same organism.

The reported instances of frank salmonellosis acquired from swimming in polluted water are minimal, but the question that remains unanswered is the amount of simple gastrointestinal upsets occurring, that might be traceable to *Salmonellae*.

OCCURRENCE AND SURVIVAL OF *SALMONELLA*

Assembled below in semi-tabular form are reported findings relative to the occurrence and survival of *Salmonella* in water.

SOURCE	FINDINGS
London Metro Board (1931; 1957-8)	In England, *Salmonella typhosa* has been isolated frequently from sewage and polluted waters since 1927. Following an outbreak of paratyphoid fever in Epping in 1931, *S. schottmuelleri* was present in large numbers in the sewage and river water. In 1958, *S. schottmuelleri* identical with the strain isolated in 1931 was still present in Epping sewage and sewage effluent. *Salmonella* were isolated

SOURCE	FINDINGS
	from 19 to 53 raw river water samples and were identified as: *schottmuelleri*, *typhimurium*, *enteritidis var*, *jena*, *anatum braenderup*, and *thompson*.
Wilson (1928; 1933; 1938)	Reported numerous isolations of *S. typhosa* from polluted waters and sewage.
Green and Beard (1938)	Recovered *S. typhosa* from 9 to 55 samples of Palo Alto sewage.
Ruchhoft (1934)	Isolated *S. typhosa* from two samples of Chicago activated sludge.
Steward and Ghosal (1938)	Isolated *S. typhosa* from the river Hooghly in India.
Mom and Schaeffer (1940)	Reported an extensive series of isolations from sewage, sludge, and river water at Bandoeng, Dutch East Indies.
Rudolfs, Falk and Ragatzkie (1950)	In their literature review on the occurrence and survival of enteric pathogens and related organisms in water, sewage, etc., these authors reported that: (1) *E. typhosa* survived in distilled or sterilized waters a few weeks to a few months, and in polluted waters and sewage only a few days, usually a week or less. That is, the survival times was in inverse relation to the degree of contamination. (2) The survival time was shorter in the summer than in the winter. Survival time varied inversely with the temperature. (3) The zone of greatest tolerance was pH 5.0 to 6.4 and increase of acid resulted in rapid mortality. (4) *E. typhosa* and other *Salmonella* survived for varying times in sewage and sludge, depending on conditions, and, in general, some of the organisms survived sewage treatment and were demonstrated in the effluent.
Messerschmidt and Wedemeyer (1951)	Reported that, at a point of discharge into the Line River, processed sewage effluent

SOURCE	FINDINGS
	contained Salmonella in 21 of 45 samples examined. Of 25 samples taken from the river up to 3 miles below the outfall, 5 contained Salmonella.
Kapsenberg (1958)	Sampled the effluent of an activated sludge treatment plant in Amsterdam from August 1956 to March 1957, using the Moore gauze-swab technique. From 3 to 9 of Salmonella were isolated from each swab. A total of 23 types was found. S. typhimurium and S. bareilly were always present, S. schottmuelleri was recovered less frequently, and S. typhosa was isolated twice.
McKinney, Langley & Tomlinson (1958)	Studied the digested sludge from a 20-day anaerobic digester and showed a 92.4 percent decrease in S. typhosa, and a 84 percent decrease after 6 days retention.
Lendon and MacKenzie (1951)	Isolated S. typhosa from water of the Wallington River, Hants, England.
Ferramola, et al (1954)	Identified 15 species of Salmonella from the Mendoza River in Argentina. Of 481 water samples examined, 31.3 percent yielded Salmonella. The predominent species were S. newport, meleagridis, oranienburg, and typhimurium.
Palazzolo, et al (1954)	Isolated 11 species of Salmonella from canal waters in the City of Mendoza, Argentina. Some 83% of the water samples taken were positive for Salmonella. The most commonly occuring species were S. newport (58.3% of samples), typhimurium, muenster, bredeney, montevideo and anatum. These six species were also isolated from children suffering from enteric disorders with the same respective frequency.
Broek and Mom (1953)	Repeatedly demonstrated S. paratyphi and S. schottmuelleri from polluted ditch waters in the community of Waalwijk, Netherlands.
Richter (1956)	Recovered S. newington, heidelberg, anatum, urbana, hull, infantio, senftenberg, and schottmuelleri from sewage-polluted channel waters in Buxtehude, Germany.

SOURCE	FINDINGS
Kraus and Weber (1958)	Found that S. typhosa survived in impounded surface water up to 26 days and S. schottmuelleri was still present after 70 days. Mortalities of bacteria were irregular and were affected by water composition and temperature.
Popp (1957)	Found that Salmonella persisted in the River Ober, Germany, for 15 to 21 miles below the point of sewage discharge.
Shrewsbury and Barson (1952)	Reported that, when stored under laboratory conditions, S. typhosa could be detected in tap water and in distilled water for 211 days and in normal saline 153 days when the initial inoculum was one billion organisms per ml.
Dunlop et al (1952)	Isolated Salmonella from 8 of 11 sewage contaminated irrigation water samples.
Dunlop (1952)	From a series of 113 samples, recovered Salmonella from 23, representing 13 species, including one S. typhosa.
Norman and Kabler (1953)	Identified Salmonella from each of 4 South Platte River water samples, from 11 of 16 polluted irrigation waters, and from 4 of 6 sewage samples. Seven different types were isolated from one sample of raw sewage, 6 types from a sample of treated sewage, and 8 types from one sample of irrigation water. The identified isolates comprised 16 types, the most prevalent being S. montevideo, typhimurium, bareilly, and newport.
Denecke (1957)	Reported the presence of S. typhosa and other Salmonellae in polluted irrigation water in Germany.
Collet et al (1953)	Were able to isolate S. typhosa from the water of a plant in Amsterdam from August 1956 to March 1957, using the Moore gauze-swab technique. From 3 to 9 Salmonella were isolated from each swab. A total of 23 types were found. S. typhimurium and S. bareilly were always present, S. schottmuelleri was recovered less frequently, and S. typhosa was isolated twice.

SOURCE	FINDINGS
Darasse, et al (1959)	Recovered 5 serotypes (*S. fann*, *ouakam*, *rubislaw*, *urbana*, and *salford*) from tap water in Dakar. A cistern reservoir at the beginning of the distribution system was contaminated by lizard droppings.

McKee and Wolf (1963) assembled the early data on the survival of *Salmonella* pertinent to water pollution (Table 21). Data such as that presented above reflect the persistent occurence in sewage-polluted surface waters of *Salmonella* species that are potentially capable of producing enteric infection in man. These organisms have been demonstrated to persist in the water for a few weeks to a few months, depending on the pH, temperature, and general condition of the water. In some cases, they have even been shown to survive sewage treatment.

SALMONELLA AND FECAL COLIFORMS

The relationship between fecal coliforms and various *Salmonella* species has been alluded to in the previous section. Although quantitative methods for estimating pathogenic bacteria in water are still in the development stage, improved analytical techniques permit isolation of *Salmonella* from waters of relatively low fecal contamination.

Data obtained by Spino (1966) indicate that *Salmonella* were consistly recovered in the Red River of the North when the fecal coliform levels were 1,000 or more. Studies on other river surveys indicate *Salmonella* could be detected occasionally for values as low as 20 fecal coliforms per 100 ml (West, 1966), and in one instance, when only four fecal coliforms per 100 ml were detected (Gallagher, 1967). The uncertain recovery of *Salmonella* below about 1,000 fecal coliforms is in part due to unpredictable *Salmonella* discharges, and in part to the sensitivity limits for the best qualitative procedures available.

APPLICATION OF METHODOLOGY TO *SALMONELLA* FACTOR

Capability (C_p) Vector

The capability of humans to resist the pathogen *Salmonella* was derived from published human dose-response test data. These tests were particularly relevant to the case of the water recreationist because the pathogens in the challenge dose were ingested orally and subjected to the action of the stomach acids. *S. typhosa* tends to produce much more severe symptoms than the other *Salmonella* and is generally credited with greater virulence. For this reason the *Salmonella* factor was divided into two groups, *S. typhosa* and other *Salmonella* - (*S.* - other), for this study.

TABLE 21

Survival Data for Salmonella (from McKee and Wolf, 1963)

Type Organisms Reported	Media and Conditions	Survival Time
S. typhosa	Spring water	10% survived 44 hours
	Synthetic well water, pH 5.9, 5°-25°C	7 days
	Synthetic well water pH 7, 9.5° & 21°C	77+ days
	Synthetic well water, pH 8, 5° & 21°C	196+ days
	Tap and distilled water, innoculum 1 X 10^9/ml	211 days
	Distilled water, innoculum 5 X 10^3/ml	59 days
	Distilled water innoculum 1 X 10^3/ml	494 days
	Water with humus added	85-104 days
	Water without humus	58-69 days
	Water or sewage, warm weather	several days
	Water or sewage, cold weather	months
	Fresh water	4 days
	Sea water	50-60% survived 44 hours
	Sea water	>30 days
	Sea water	>32 days
	Autoclaved sea water	several days

TABLE 21 (continued)

Type Organisms Reported	Media and Conditions	Survival Time
S. typhosa (cont.)	Polluted water	>4 days
	Shell oysters	14-60 days
	Within gut of Nematodes of Rhabditidae family	3-4 days
E. typhosa	Sea water, heat sterilized	25 days
	Sea water, raw	9 days
	San Francisco Bay 10°C	12-28 days
	San Francisco Bay filtered, 10°C	14-34 days
	Tidal water	>2-3 weeks
	Sea water	14 days
	Imhoff tank sludge	11 days
S. paratyphi	Spring water	10% survived 44 hours
	Sea water	50-60% survived 44 hours
S. paratyphi A	Tap water, aged 24 hours	29 days
S. paratyphi B	Surface water	3 months
	Autoclaved sea water	several days
	Sea water, innoculum 1×10^7 /ml	2 months
	Tap water, aged 24 hours	25% remaining in 29 days

TABLE 21 (continued)

Type Organisms Reported	Media and Conditions	Survival Time
S. schottmuelleri	Sludge banks and ditch water	1.5 years
	Oysters 5°C	49 days
S. enterifides	Spring water	10% survived 44 hours
	Sea water	50-60% survived 44 hours
S. typhiabdominalis	Tap water, aged 24 hours, + 0.2gm/l sterilized feces, 16°-18°C	multiplication
S. typhimurium	Sea water	few died in 24 hours
Salmonella	Soil and potato (surface)	40+ days
	Carrot (surface)	10+ days
	Cabbage and Gooseberries (surface)	5+ days
	Muddy waste water	130 days
Typhoid bacteria	Water, 0°C	9 weeks
	Water, 5°C	7 weeks
	Water, 10°C	5 weeks
	Water, 18°C	4 weeks
	Tap, well and distilled water	2 weeks to 80 days
	Sea water	12-16 hours
	Septic sewage	5 weeks
	Excreta	10-84 days
	Septic tanks	>27 days

TABLE 21 (continued)

Type Organisms Reported	Media and Conditions	Survival Time
Typhoid bacteria (cont.)	In gut of fish	7-9 weeks
	Carp	4-6 weeks
	Oysters	9-42 days
Paratyphoid bacteria	Sea water	21 days

Salmonella Typhosa C_p

The dose response test data on human volunteers published by Hornick, et al, is the most complete and quantitatively reliable body of data dealing with this subfactor. In these studies, human volunteers were inoculated with graded doses of *S. typhosa* and the frequency of infection (illness) noted.

Based on these studies, a histogram was constructed in which each step of the histogram represented an average of the data points of volunteers ill at each concentration of challenge dose (the details of this analysis and a discussion of some of the essential assumptions is presented in Appendix B).

Twenty-five pseudo-random numbers were generated, and samples were obtained from the histogram. These samples were input to the BSTFIT program. The results of this analysis are shown in Table 22. This table represents the maximum likelihood estimates of the parameters describing the normal, lognormal, exponential, Weibull, and Gamma distributions. Based on the Kolmogorov-Smirnov goodness "or" fit (or "d") test, it can be established that the lognormal probability density function is the best description of the universe from which the dose response sample was drawn.

Figure 13 shows the lognormal distribution selected by BSTFIT, along with the upper and lower 95% confidence intervals.

Salmonella - other, C_p

The *S.* -other C_p density function is based upon work published by McCullough and Eisele in 1951 dealing with strains of *S. bareilly*, *S. newport*, *S. anatum* I, II, and III, *S. meleagridis* I, II, and III, *S. derby*, and *S. pullorum* I, II, III, and IV. Thus groups of the Kaufman-White Scheme B, C_1, D, and E, the majority of the groups significant to humans, were covered. As in the case of *S. typhosa*, the pathogens were fed to the volunteers and thus the results are directly applicable to the case of recreation water quality. These tests were performed in a way to emphasize the detection of a "minimum effective dose." They were conducted in a manner quite similar to "step stress testing to malfunction." Thus the dose response data could be input to BSTFIT directly without recourse to sampling from a histogram.

The results of the analysis are shown in Table 23. Once again the lognormal probability density function has been selected at the 5% level of significance. This probability density function is shown graphically in Figure 14, along with its upper and lower 95% confidence limits. Appendix B presents the complete analysis.

The BSTFIT analysis of the available *Salmonella* data challenge some commonly held concepts on *Salmonella* infectivity. The idea that *S. typhosa* is much more infectious than the other *Salmonella* species is seriously challenged. The percent of population becoming ill can be compared for the two groups of *Salmonella*.

TABLE 22

S. typhosa Capabilities Vector (C_p)

Using 25 Random Numbers

(Input Data)

Name of distribution	"d" Statistic	Probability that data came from cited distribution
Normal	0.538	0 +
Lognormal	0.191	0.317
Exponential	0.936	0 +
Gamma	0.2981	0.0235
Weibull	-	-

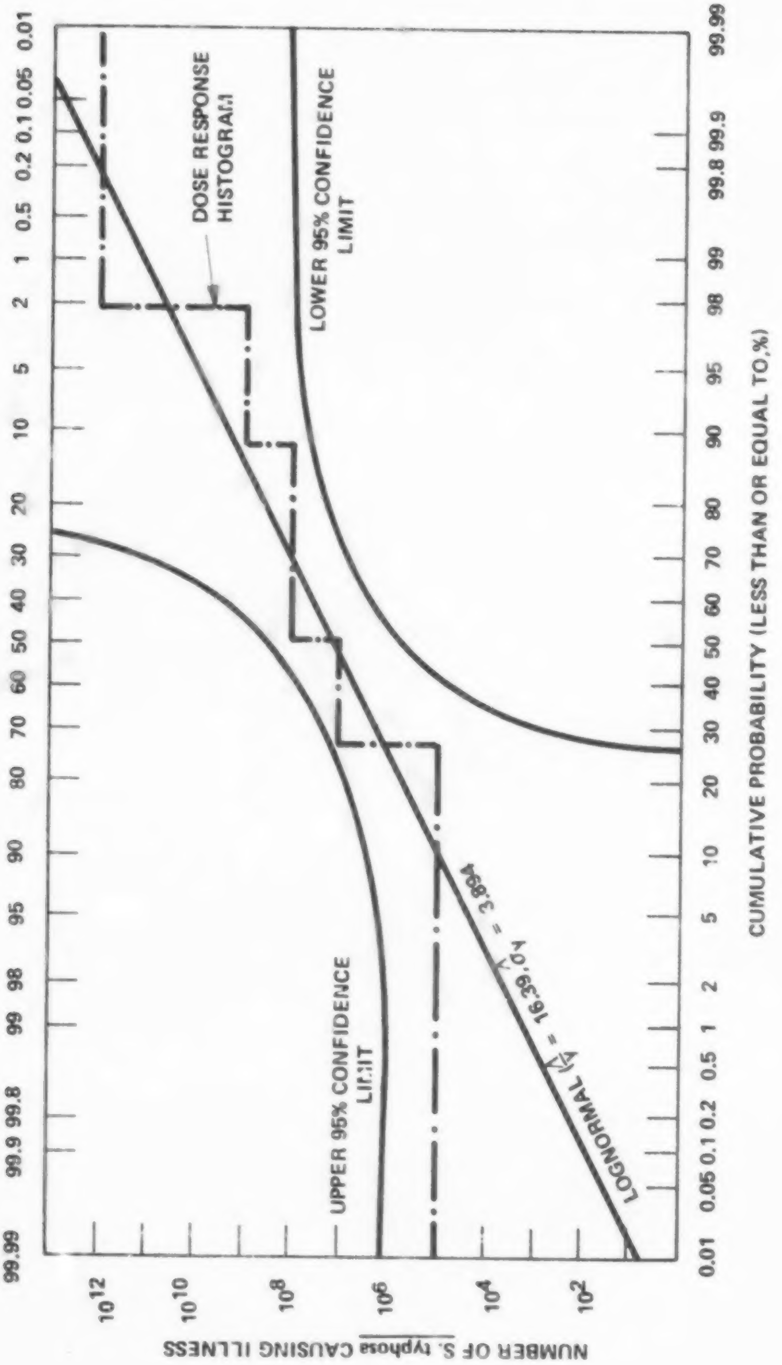


Figure 13. S. typhosa Capabilities Vector (C_p)

TABLE 23

S. -other Capabilities Vector (C_p). for 69 Challenge Cases

Name of distribution	"d" Statistic	Probability that data came from cited distribution
Normal	0.3926	0 +
Lognormal	0.1636	0.0497
Exponential	0.6589	0 +
Gamma	0.2818	0.0000348
Weibull	0.5578	0 +

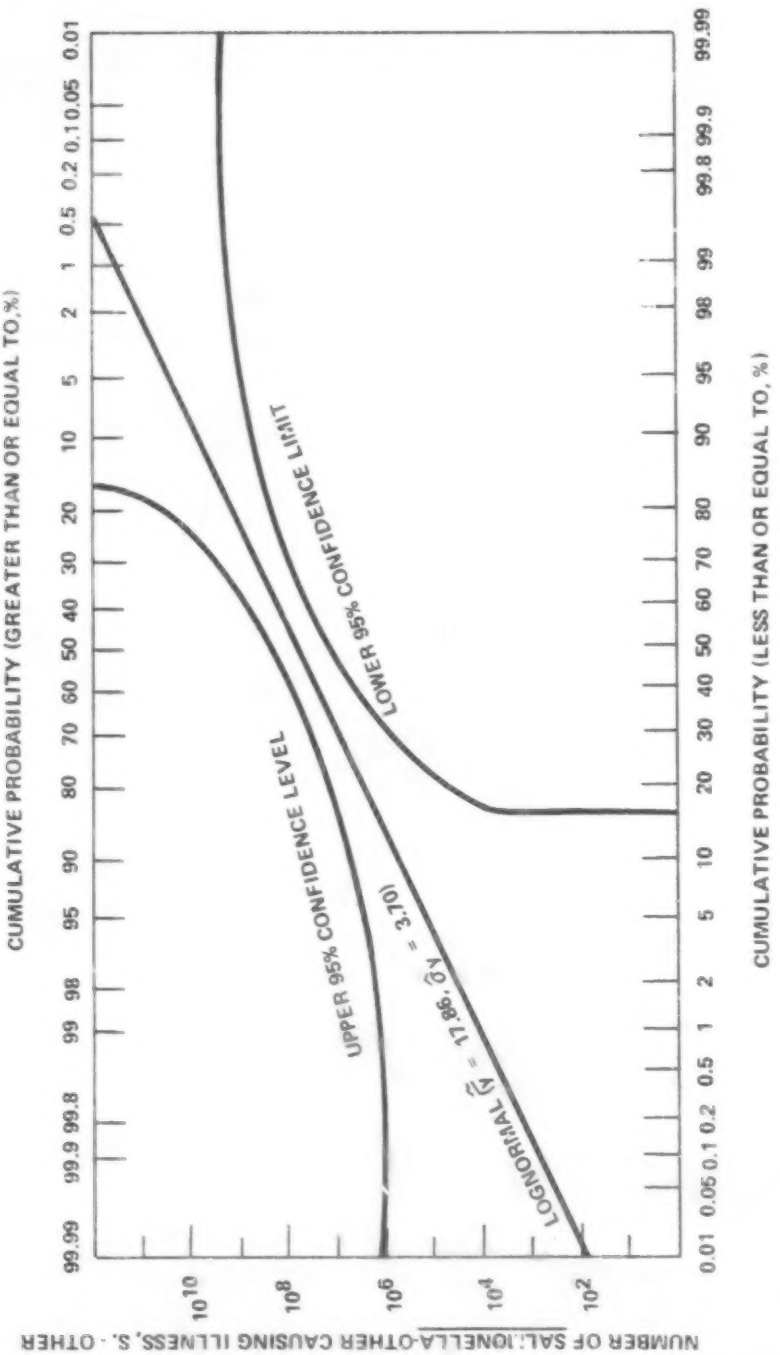


Figure 14. Salmonella - Other Capabilities Vector (C_p)

Population Becoming Ill %	Organisms Ingested	
	S. typhosa	S. -other
50	8×10^6	5×10^7
10	1×10^5	5×10^5
1	1×10^3	1×10^4

It is obvious from the comparison that reports stating that one to ten S. typhosa organisms constitute an infective dose are unrealistic (Kehr, 1943).

A more important result of this study is the similarity of dosages for the two Salmonella groups, indicating that, in general, infectivity for the various Salmonella is rather similar. This will permit handling the entire Salmonella factor as a single group in future analysis.

Requirements (R_q) Vector

The formulation of the Requirements (R_q) Vector for the Salmonella requires quantitative measurements on the distribution of the pathogens in the waters of interest. The probability distribution of Salmonella in a water would be evaluated for the likelihood that a recreationist would encounter a given number of Salmonella while in the water.

It is remarkable that nowhere in this country are the number of Salmonella per volume of water routinely counted and indeed it is almost impossible to uncover any estimate of Salmonella numbers in natural waters of any type. In almost all cases where this factor is measured, the results are determined in binary form (presence or absence of Salmonella), and sometimes additional studies are performed to identify the strain(s) present.

A study representing estimates of Salmonella concentration in an estuary was conducted by McCoy (1964) in England. His findings are reproduced here as Table 24. It will be noted that these results are categorized in terms of MPN (most probably number) per liter, a form sometimes used to report the presence of coliform bacteria. The use of binary tests to predict the number of coliform bacteria in a sample has been discussed in the literature for many years. The validity of the methodology rests on the favorable comparison of test results using known or true mean densities inoculated into appropriate fermentation tubes and the MPN predictions calculated from the number of tubes showing positive growth.

The numbers shown in Table 24 may be very low. Bucyowska (McCoy, 1964) indicates that quantitive measurements in Polish coastal waters

TABLE 24

Salmonella Factor Requirements Data
(from McCoy, 1964)

MPN org/liter	No. of Samples	Percent Positive	Cum. Percentage
0	152	38.8	38.8
1-9	85	21.6	60.4
10	66	16.8	77.2
20	35	8.9	86.1
35	13	3.3	89.4
50	9	2.3	91.7
70	8	2.0	93.7
90	6	1.5	95.2
120	4	1.0	96.2
161	5	1.3	97.5
230	3	0.8	98.3

showed that "... a large number of these samples contained 1,000 *Salmonella* per litre." In the 392 samples examined by McCoy, 240 or 61.2% had at least 1 MPN/liter *Salmonella*.

Consideration should also be given the fact that saline water *Salmonella* recoveries are consistently lower than in fresh water and that the percent recovery using various enrichment media that depend on a selective toxicant (e.g., tetrathionate) has not been worked out. However, it is quite likely that the relative distribution of sample count versus percent positives reported by McCoy is an accurate estimate and that on this basis a probability distribution can be carried out. Accordingly, the numbers in the table were scaled up by factors of 10^3 and 10^5 to bring the MPN count up to a range that could be expected to be present during an actual outbreak.

Next, it was assumed that a recreationist will ingest 10 ml of water (Streeter, 1951), although there is really no statistical basis for this assumption; because it does seem to be a conservative estimate, the MPN numbers were again adjusted upward by an order of magnitude.

However, as discussed earlier, the C_p fits had inherent in their histograms the assumption that the cited challenge doses was actually the lower end of the class interval (see Appendix B). Since this assumption could be considered unconservative, the R_q data were increased by two orders of magnitude.

In summary, then:

$$\underline{S.}\text{ - total }(R_q) = \text{MPN }(\underline{\text{Salmonella}})/\text{liter} \times 10\text{ ml ingested}$$

$$\times \frac{1\text{ liter}}{1000\text{ ml}} \times 100$$

(for compatibility with C_p data)

When more information is made available on the requirements vector, some of these assumptions may prove to be unnecessary.

Just as in the C_p analysis, a histogram was sampled using 25 random numbers and the sample input to BSTFIT. The Kolmogorov-Smirnov goodness of fit test selected (Table 25) a Weibull as the preferred distribution. It should be noted that all four maximum likelihood best fits were very poor, reflecting a significant lack of confidence in the data. The improvement in the accuracy of measurement of the requirements vector is an area in which fruitful research can and should be performed.

The BSTFIT density function for the *Salmonella* factor was converted to *S. typhosa* and S.-other by ratioing the *Salmonella* R_q density function by 5% and 95% respectively, based on the frequency of isolation of the various species (Moore, 1900). The results of the convolution analysis, as discussed in the next section, showed that the subdivision of the factor *Salmonella* into *S. typhosa* and *S.*-other was a needless refinement.

TABLE 25

Salmonella Total Requirements Vector (R_q)
Using 25 Random Numbers

Name of distribution	"d" Statistic	Probability that data came from cited distribution
Lognormal	0.364	0.00265
Exponential	0.360	0.00307
Gamma	0.359	0.00317
Weibull	0.359	0.00319

The two probability distributions, dose effect and factor concentration, were used to develop the basic risk curve by determining their interaction. The basic convolution was then truncated to develop the risk versus factor concentration criteria.

Truncation of the basic curve - equivalent to criteria - reduces the risk as the R_q cut-off point approaches the mean. In Table 26 the basic untruncated convolution can be seen to yield a probability (risk) of illness of 0.000423 (4/10,000) for *S. typhosa* and 0.001208 (1/1,000) for *S.*-other. This indicates that there is only a small difference, if any, in the infectivity of the two *Salmonella* groups.

The results of the convolutions carried out on the two groups of *Salmonella* using the two different R_q sets (McCoy x 1,000 and McCoy x 100,000) were examined to determine the relationships between the two *Salmonella* sub-factors. This is illustrated in Figure 15.

The equation of the risk-concentration relationship was next developed. Attempts were made to fit both decimal and logarithmic functions to the risk-concentration data and from the analysis of variance to select the most probable fit. The linear regression was selected as the most descriptive polynomial and this form, presented in Table 27, was used in preparing the dose vs risk curve in Figure 15, along with the 50% confidence limits.

The wide spread in the data as compared to the virus analysis indicates that more work is required on this factor. The most probable area of uncertainty is in the frequency distribution of the *Salmonella* concentration in the water (R_q vector). The analysis was based on only one

TABLE 26

Truncated Convolution of Dose Effect and Factor Concentration Based on McCoy's Data (x 1000)

S. typhosa				***S.*-other**			
CONVOLUTION $(C_p - R_q)$		CRITERIA (Truncation point of R_q organism/l	P (Illness)	CONVOLUTION $(C_p - R_q)$		CRITERIA (Truncation point of R_q organism/l	P (Illness)
Basic	Basic	None	0.000423	Basic	Basic	None	0.001208
↓	$\bar{x} + 5\sigma$	6,079	0.000233	↓	$\bar{x} + 5\sigma$	115,391	0.000809
	$\bar{x} + 4\sigma$	4,982	0.000192		$\bar{x} + 4\sigma$	94,665	0.000691
	$\bar{x} + 3\text{-}1/2\sigma$	4,437	0.000172		$\bar{x} + 3\text{-}1/2\sigma$	84,303	0.000628
	$\bar{x} + 3\sigma$	3,891	0.000151		$\bar{x} + 3\sigma$	73,940	0.000561
	$\bar{x} + 2\text{-}1/2\sigma$	3,346	0.000131		$\bar{x} + 2\text{-}1/2\sigma$	63,578	0.000492
	$\bar{x} + 2\sigma$	2,801	0.000110		$\bar{x} + 2\sigma$	53,215	0.000419
	$\bar{x} + 1\text{-}1/2\sigma$	2,255	0.000089		$\bar{x} + 1\text{-}1/2\sigma$	42,852	0.000343
	$\bar{x} + 1\sigma$	1,710	0.000068		$\bar{x} + 1\sigma$	32,490	0.000265
	$\bar{x} + 1/2\sigma$	1,165	0.000046		$\bar{x} + 1/2\sigma$	22,127	0.000183
Basic	$\bar{x}$	619	0.000025	Basic	$\bar{x}$	11,764	0.000099

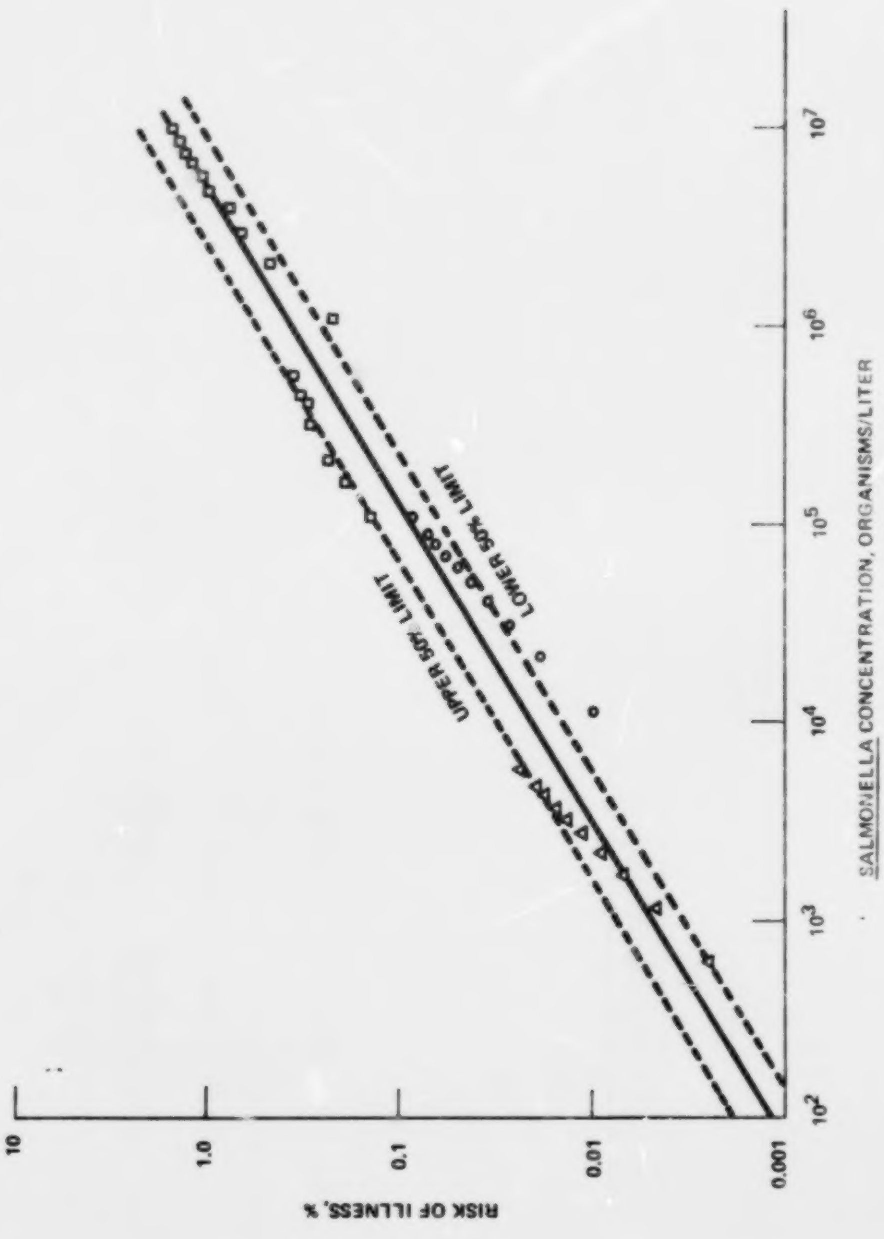


Figure 15. Criteria for the Presence of Salmonella in a Recreational Water

TABLE 27

Derivation of Equation to Describe Recreationist Risk as a Function of Salmonella Criteria

Equation of polynomial best fit	Analysis of variance		
	F* Calculated	F Minimum allowable from table**	
		Level of significance	
		5%	1%
$\mathrm{Log}_e\ Y = -14.1365 + 0.61049\ \mathrm{Log}_e\ X$	1,037	4.12	7.42

---

[*] F distribution, which is the distribution of the ratio of two variances.

[**] From tables of critical values of F at 5% and 1% levels. When the calculated value for F exceeds the critical value, the answer is significant.

study of *Salmonella* distribution (McCoy, 1964) and several assumptions had to be made before this data could be used. The need for additonal surveys is strongly emphasized. The lumping of the *S. typhosa* dose-response data with the other *Salmonella* may also contribute to the lower confidence levels. However, until more distribution data is available, this view can only be regarded as being speculative.

The analysis is nonetheless important in that it does quantify the risk to a population using a given water contaminated with *Salmonella*. The risk of ill effect of 2 cases/10,000 population at a count of 10,000 *Salmonella* organisms per liter does not appear unreasonable. There is now sufficient information on hand to justify considering criteria based on estimating the actual number of *Salmonella* in a recreational water.

An analysis of the *Salmonella* data similar to that carried out for the viruses can be prepared. This would demonstrate the application of the methodology to control risk in a population. If one case of illness per 100,000 population is selected as the standard, the concentration requirements for *Salmonella*, coliform, and fecal coliform can be determined.

In this case, the estuary studied by McCoy will be used as the example. From the convolution of the dose-response data and the factor distribution it is determined that a concentration of 80 MPN *Salmonella*/liter constitutes a 1/100,000 risk of illness. In this case, the *Salmonella*-coliform ratio is derived from actual studies carried out in the estuary by McCoy (1964). This relationship is presented in Figure 16 below the *Salmonella* concentrations and below this the fecal coliform-coliform relationships developed by Strobel (1968) are also presented. From the figure we can conclude that a risk 1/100,000 for developing illness due to *Salmonella* is probable when counts of 8×10^5 MPN coliforms/100 ml or 6.9×10^5 MPN fecal coliforms/100 ml are present at the various sampling points.

The State standards of 5000 MPN coliform/100 ml presents less than $1/1 \times 10^6$ risk of illness, which is clearly negligible. This could explain the almost complete lack of epidemiological evidence for *Salmonella* infections even when recreationists use heavily polluted waters (Moore, 1954; McCoy, 1964; Flynn, 1965). In the two analyses presented it is obvious that the virus criteria will dominate and serve as the basis for standards setting.

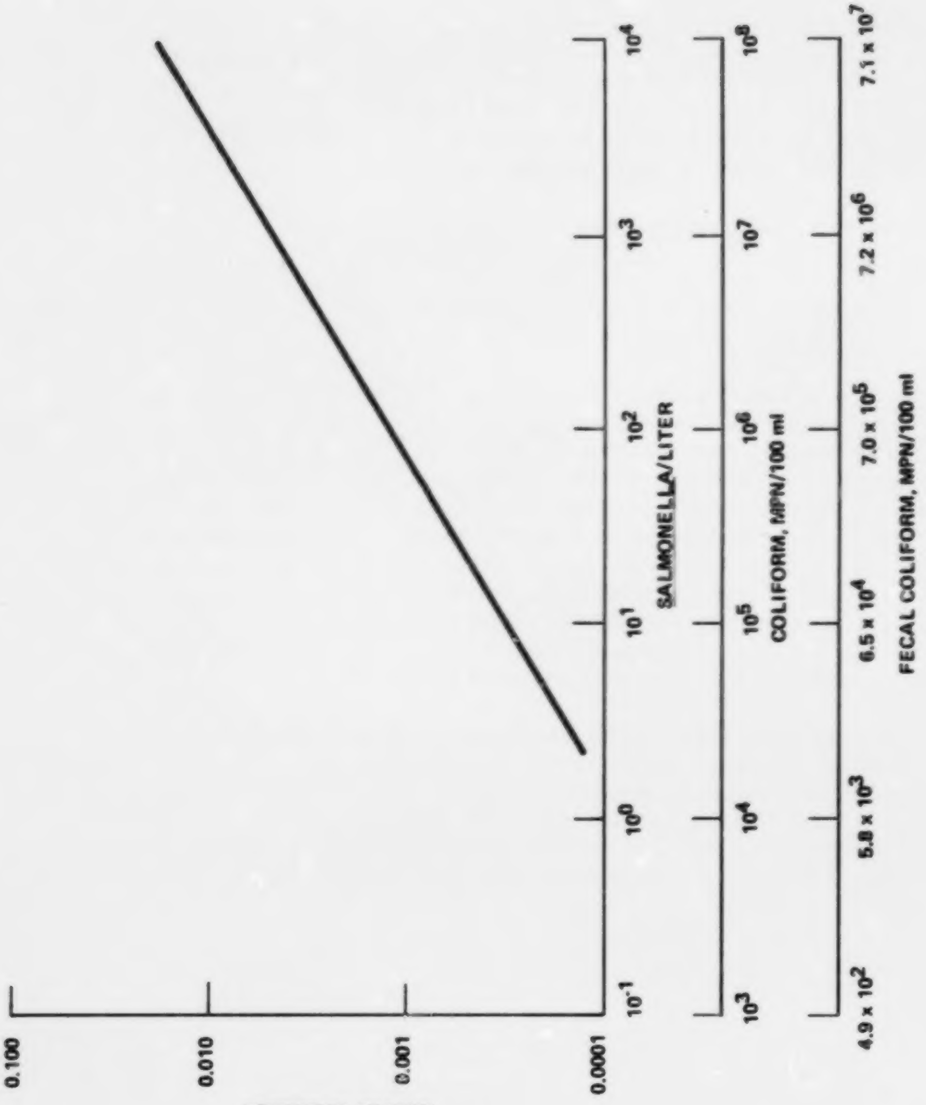


Figure 16. Coliforms and Fecal Coliforms as Indicators of Salmonella Risk

SECTION VIII

TOTAL AND FECAL COLIFORM AS INDICATORS OF RECREATIONAL WATER QUALITY

GENERAL CONSIDERATIONS

The fecal coliform test to detect pollution by warm-blooded animals has been proposed as a better bacteriological measurement of public health hazards in stream pollution investigations, in sewage treatment systems, and in bathing water than the traditional total coliform bacterial procedure. The fecal coliform is gradually gaining acceptance as a routine supplementary determination (Geldreich, 1967).

THE BACTERIA OF INTEREST

Most bacteria in water are derived from contact with air, soil, living and decaying plants or animals, and the fecal excrement from warm and cold-blooded animals. Present interest is in those species or groups that can be considered a hazard to public health.

The pollution of water with fecal matter may add a variety of intestinal pathogens having the potential of causing human or zoonotic disease.

While it is recognized that harmful contamination of outdoor bathing beaches may be caused by sewage, by the dumping of refuse, or by the individual bathers themselves, the transmission of disease by bathing waters has not been established as a major public health hazard (Stevenson, 1953; Moore, 1959). The condition of bathing waters varies from day to day and season to season, and it may be altered by:

(1) Fixed sources of pollution

(2) Incidence of disease among the local population

(3) The bathing load

(4) Physical factors, such as wind, weather, and temperature

(5) Composition of the water

Other important questions must be answered, including:

(1) How important is the small amount of water swallowed while bathing as an etiological factor?

(2) Which diseases are transmissible by bathing water?

(3) To what degree are diseases contracted at beaches through exposure to infected water, infected people, and infected appurtenances?

Pathogens

The most common genera of pathogenic organisms found in water are *Salmonella*, *Shigella*, *Vibrio*, *Mycobacterium*, *Pasteurella*, and *Leptospira*, as well as certain enteric and respiratory viruses. Although these pathogens have been found, a wide variety of media and methods are necessary to detect them. Recent studies of *Salmonella* detection in sewage (Dunlop, 1957), irrigation water (Dunlop, et al, 1951; 1952), river water (Spino, 1966) and tidal waters (Brezenski, et al, 1965) produced promising techniques that permit the isolation of these pathogens from waters with relatively low coliform densities. For example, *Salmonella* were isolated from the Red River of the North when the coliform density was 2,200/100 ml and the fecal coliform level was 220/100 ml. According to Geldreich (1966), the use of these organisms as pollution indicators may not be desirable. The intervals between sampling, pathogen detection, and condemnation of a water supply would result in an exposure risk to the consumer or recreationists; moreover, failure to demonstrate pathogenic organisms does not always ensure a safe water.

Indicator Organisms

Studies on the origins of fecal coliforms and fecal streptococci have generated renewed interest in these groups as better indicators of pollution by warm-blooded animals than the traditional coliform procedure used in water pollution studies.

To be of value, a biological indicator of contamination or pollution must satisfy the following criteria (Fair and Geyer, 1956; McCarthy, 1961):

(1) It must be a reliable measure of the potential presence of specific contaminating organisms, both in natural waters and in waters that have been subjected to treatment. To meet this requirement, the indicator organism or organisms must react to the natural aquatic environment and to treatment processes, including disinfection, in the same way, relatively, as do the contaminating organisms.

(2) It must be present in numbers that are relatively much larger than those of the contaminating organism whose potential presence it is to indicate. Otherwise, detection of the contaminating organism itself would serve a more useful purpose.

(3) It must be readily identified by relatively simple analytical procedures.

(4) It must lend itself to numerical evaluation as well as qualitative identification.

The Total Coliform Group

Coliform bacteria have traditionally been the bacteriological tool used to measure the occurrence and intensity of fecal contamination in water pollution investigations for nearly 60 years, largely because they meet the criteria outlined above. During this time, a mass of data has been accumulated to permit a full evaluation of the sensitivity and the specificity of this bacterial pollution indicator.

As defined in Standard Methods for the Examination of Water and Wastewater (APHA, 1965), "the coliform group includes all of the aerobic and facultative anaerobic, Gram-negative, non-spore-forming, rod-shaped bacteria which ferment lactose with gas production within 48 hours at 35°C." From this definition, it becomes immediately apparent that this bacterial grouping is somewhat artificial in that it embodies a heterogeneous collection of bacterial species, having only a few broad characteristics in common. Yet for practical applications to surface water pollution studies, this grouping of selected bacterial species, termed the total coliform group, has proved to be a workable arrangement.

The total coliform group merits consideration as an indicator of pollution because these bacteria are always present in the normal intestinal tract of humans and other warm-blooded animals and are eliminated in large numbers in fecal wastes. Thus the absence of total coliform bacteria is evidence of a bacteriologically safe water.

The relationship between the bacterial quality of bathing water and the incidence of illnesses in swimmers, as compared with non-swimmers, was studied by Streeter (1951). His analysis is based on:

(1) The incidence of typhoid and paratyphoid in a region

(2) The morbidity - mortality ratio

(3) The relationship between these diseases and other enteric infections

(4) The ratio of coliforms to pathogens

(5) The frequency of swimming

(6) The assumption that 10 ml of water will be swallowed by each bather each day.

(7) The probability that this ingestion will cause illness

Streeter (1951) used the findings of Kehr and Butterfield (1943) as a basis for an analysis of the rationality of various proposed bathing water standards, as viewed from the standpoint of water-borne disease hazards. Kehr and Butterfield (1943) reviewed a number of studies in England, Indonesia, and California, where the successful enumeration of both coliforms and typhoid and para-typhoid organisms was carried out in sewage and sewage-polluted waters at the time of outbreaks of these enteric diseases. They derived a correlation between the morbidity rates from typhoid fever in different areas and the ratios of E. coli to E. typhosa in the sewage and sewage-polluted waters of the areas (see Figure 17).

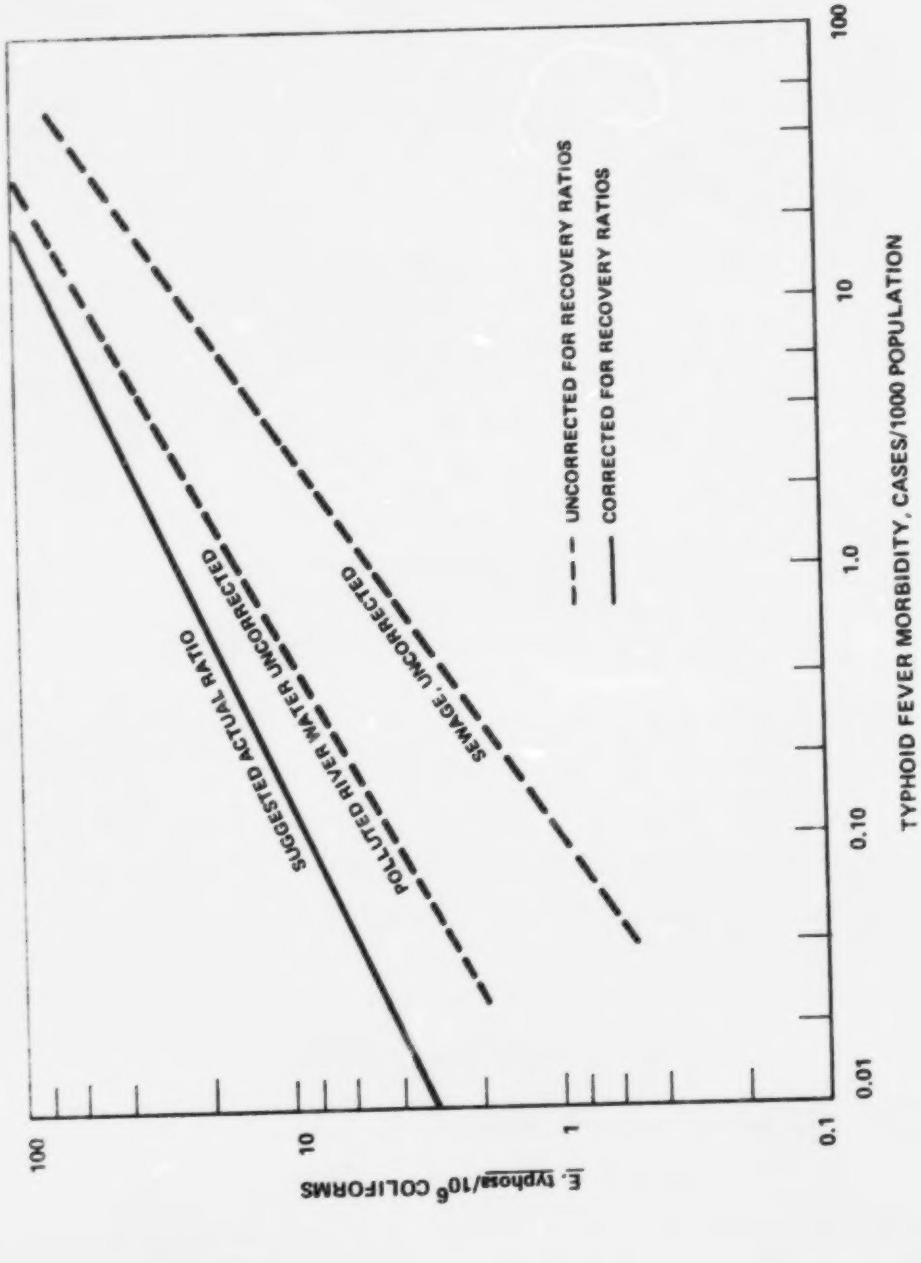


Figure 17. E. typhosa per Million Coliforms for Varying Typhoid Fever Morbidity Rates

The following account is an extract of Streeter's study on bacterial objectives for the Ohio River, and is quoted because the reasoning contained therein has indirectly formed the basis for most, if not all, of the state total coliform standards.

"According to U.S. Census mortality reports for various diseases, the average typhoid mortality rate for seven Ohio River states in the years 1945-47 was 0.4 per 100,000 (as compared with a rate of 0.2 per 100,000 in the U.S. Registration Area). Assuming a morbidity:mortality ratio of 10 to 1, this would indicate a morbidity rate of 4 per 100,000, or 0.04 per 1000." From Kehr and Butterfield's curve, the corresponding ratio of *E. typhosa*:*E. coli* in the sewage and sewage-polluted waters of such an area would be about 170,000 coliforms for each *E. typhosa* organism. This, of course, is an extremely low infection ratio for typhoid fever, but nonetheless measurable according to the Kehr-Butterfield results.

In order to apply these data to an evaluation of the typhoid hazard in the bathing waters of an area, it is necessary to assume the average volume of water ingested per bather per day. For the purposes of estimate, let this volume be assumed as 10 ml, which probably would be high for trained swimmers, and low for children.

Now let:

R = The number of coliforms per single *E. typhosa* in the bathing water

B = The number of bathers per day

V = The volume of water, in ml ingested per bather per day

C = The average coliform content of the bathing water per ml

Then, the chance of exposure (P_e) of (B) bathers to a single *E. typhosa* on any day is:

$$P_e = BVC/R$$

and the exposure interval, in days, between successive ingestions of a single organism is:

$$I_e = 1/P_e = R/BVC$$

For illustration, let us assume R = 170,000; V = 10 ml, and C = 10 per ml or 100 per 10 ml. Then the chance that a single bather would be exposed to ingestion of one *E. typhosa* organism would be:

$$P_e = 1/1,700$$

During a 90-day bathing season, *if he bathes every day*, his risk of exposure will be 90/1700, or 1/19.

Kehr and Butterfield estimated that about two percent of persons exposed to ingestion of a single E. typhosa organism actually contract the disease. On this basis, it may be estimated that the bather's risk of contracting typhoid fever during a 90-day season would be 1/19 x 1/50, or 1/950.

A fundamental assumption here is the estimate that one E. typhosa ingested will infect two percent of the exposed subjects. Based on the dose response curve (Figure 13) developed from actual S. typhosa ingestions by human volunteers it is apparent that more than 1000 S. typhosa must be ingested to give a two percent probability of illness. Because the two types of typhosa are of roughly the same virulence, the early work of Kehr and Butterfield, probably overestimates the infectivity of E. typhosa.

EPIDEMIOLOGICAL STUDIES ON HEALTH/WATER QUALITY RELATIONSHIPS

Epidemiological and statistical studies of populations that have been bathing or swimming in contaminated waters provide a direct approach to assessing recreational water quality. The number of such investigations has been limited to only three in the United States. A study of this type was made for the U.S. Public Health Service by Stevenson et al (1953). These workers undertook a series of field studies of selected population groups swimming in waters of different bacterial quality to determine the relationship between incidence of illness among swimmers and the coliform density of the water. Table 28 summarizes some of the basic data collected.

The results indicate that illness occurrs more frequently among swimmers than non-swimmers. This observation is not surprising in view of the fact that water is an abnormal habitat for man, regardless of its quality. The results also showed that when total illnesses among swimmers and non-swimmers were compared, except as noted below, there appeared to be no significant correlation between illness incidence and quality of the water in the areas studied.

It might also be of interest to note that among swimmers, eye, ear, nose, and throat ailments represented more than half of all the illnesses recorded, gastrointestinal disturbances about 20%, and skin irritations the remainder. Eye, ear, nose, and throat ailments represented an even higher percentage, 68 percent, of pool-swimmer illnesses.

Stevenson et al (1953) actually found a specific correlation between illness incidence and quality of water in two instances. In the first case, rates were measured for several days following three-day periods of high and low bacterial concentrations at one beach on Lake Michigan. It was observed that illness frequency was significantly higher among swimmers when the water had an average coliform density of 2300 per 100 ml than when the average density was 43 per 100 ml.

In the second instance, swimming in the Ohio River when the "median coliform density" was 2700 per 100 ml appeared to have caused a significant increase in gastrointestinal illness, although the total number of illnesses was small (Smith and Woolsey, 1951).

TABLE 28

Summary of Data from Studies of Bathing Water Quality

(after Stevenson, 1953)

		Coliform Concentration, MPN per 100 ML			No. of Illnesses per 1000 Person-Days	
Water Body	Location	Median	Minimum	Maximum	Among Swimmers	Among Non-Swimmers
Lake Michigan	Chicago I	91	9.1	3,500	7.1	3.7
Lake Michigan	Chicago II	190	23	24,000	8.3	5.6
Ohio River	Kentucky	2700	230	160,000	8.8	7.4
Pool	Kentucky	<3	-	-	13.8	-
Tidal Water	New Rochelle, N. Y.	610	<30	460,000	5.3	3.3
Tidal Water	Mamaroneck, N. Y.	253	<30	460,000	6.2	3.3

Stevenson et al (1953) point out that these two cases do not constitute conclusive evidence of correlation between illness and bathing water quality, because the numbers of individuals and days involved were so few.

A subsequent study was conducted by USPHS personnel on Long Island Sound to determine the relationship between illness and bodily exposure to contaminated salt water.

This investigation gave no evidence that variation in water quality of the sort encountered is capable of producing marked differences in the amount of illness experienced by swimmers. The data from this study were of sufficient internal consistency to indicate that significant effects would have shown up had they existed.

An intensive epidemiological investigation was conducted by Moore, and his committee on bathing beach contamination, in England and Wales (1954; 1959). Extensive bacteriological and epidemiological studies were made over a period of five years in relation to more than 40 popular bathing beaches, the waters of the great majority of which were subject to contamination with sewage. The median presumptive coliform counts varied from 40 to 25,000 per 100 ml; as many as 40 percent of the samples contained over 10,000 coliforms per 100 ml. In addition to coliform bacteria, determinations were made for members of the Salmonella group, of which 33 different species were isolated.

The general conclusions of the Moore committee were that bathing in sewage-polluted water carries only a negligible risk to health, and, where the risk is present, it is probably associated with chance contact with intact aggregates of infected fecal material. In the entire study, there were only four cases of paratyphoid fever that could possibly have been attributed to bathing in infected sea water. In each case, however, the bathing area was contaminated with visible fecal material. The committee indicated that unless the water is so fouled as to render the bathing beach esthetically revolting, it would seem that public health requirements are reasonably well met by the present British policy of improving grossly unsanitary beaches and preventing as far as possible the pollution of the waters with undisintegrated fecal matter. The extreme view of the British workers is not shared by American public health authorities.

In order for the coliform test to continue as a useful criterian of health risk from polluted waters, it must be quantitatively related to the presence of pathogens. This requires that the relative distribution of pathogens and coliforms in a given water be determined and a correlation calculated. The basic data required to carry out this analysis was fortunately available from the studies of McCoy (personal communication). He simultaneously estimated that numbers of coliforms and numbers of Salmonella present in an estuary over a given period of time. This information is presented in Table 29. The data and the calculated regression line are presented in Figure 18. The equation for the line, in this case a quadratic regression, was determined to be:

TABLE 29

Occurrence of *Salmonella* and *E. coli* in an Estuary

(MPN *Salmonella* and *E. coli* I ($x10^{-6}$) Normalized for Flow of 1 MGD)

Date		Time				MGD Correction
		1000-1500	1600-2100	2200-0300	0400-0900	
Aug 1/2	*Sal.*	9.96	6.66	<1.66	<1.66	$\left(\frac{1}{0.60}\right)$
	E. coli I	1.83	<0.299	0.299	0.299	
8/9	*Sal.*	1.58	1.58	<1.58	<1.58	$\left(\frac{1}{0.63}\right)$
	E. coli I	1.10	2.85	>28.5	0.634	
15/16	*Sal.*	1.96	22.5	9.8	9.8	$\left(\frac{1}{1.02}\right)$
	E. coli I	0.176	0.176	>17.6	0.176	
22/23	*Sal.*	14.7	14.7	8.82	1.47	$\left(\frac{1}{0.68}\right)$
	E. coli I	2.65	1.62	1.02	0.59	
29/30	N. G.					$\left(\frac{1}{0.68}\right)$
Sept 5/6	N. G.					$\left(\frac{1}{0.62}\right)$
12/13	*Sal.*	>164.	>164.	98.3	37.7	$\left(\frac{1}{0.61}\right)$
	E. coli I	29.5	11.35	2.93	11.35	
19/20	*Sal.*	25.6	38.5	6.41	38.5	$\left(\frac{1}{1.56}\right)$
	E. coli I	1.15	7.05	0.256	4.44	
26/27	*Sal.*	1.78	3.57	3.57	1.78	$\left(\frac{1}{0.56}\right)$
	E. coli I	1.96	1.96	1.23	1.23	
Oct 3/4	*Sal.*	<1.66	1.66	1.66	<1.66	$\left(\frac{1}{0.60}\right)$
	E. coli I	< 0.30	0.666	0.666	0.30	

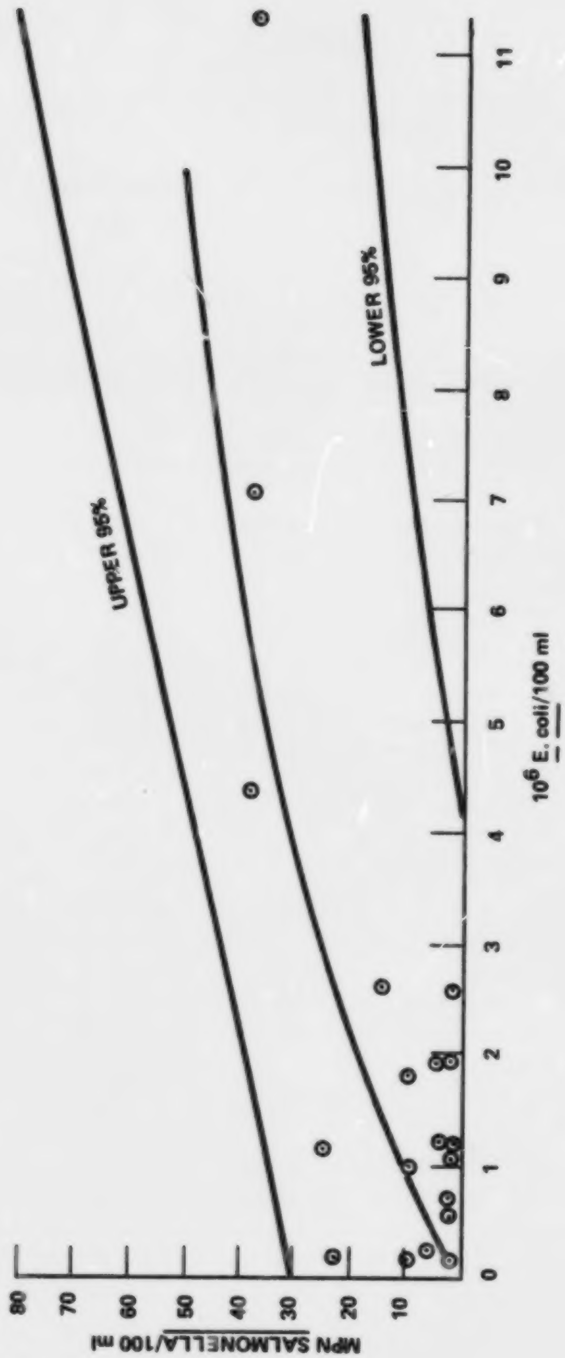


Figure 18. Relationship of Number of Salmonella to Coliform in an Estuary

$$Y = -4.1 \times 10^{-2} + 1.07 \times 10^{-5} X - 6.55 \times 10^{-3} X^2$$

This equation was used to calculate the relationship at low levels of coliform number and the expanded lower portion of the curve is presented in Figure 19. The scatter of the data at the low end of the curve prevented a more accurate estimate of the slope and it was not possible to bring the cuve through the origin.

The curve in Figure 19 predicts a concentration of 10 Salmonella organisms/100 ml for an MPN of 1×10^6 coliform/100 ml, a figure that is not too different from Kehr and Butterfield's estimate of 6 S. typhosa/10^6 coliforms. This information was then superimposed on the risk curve for Salmonella shown in Figure 16.

Based on the above analysis, it is concluded that the criteria for total coliform, as an indicator of health risk, have in the past been very conservatively estimated. It is of little wonder that the value and reliability of the coliform test, as an indicator and predictor of disease hazards in recreational, has been seriously questioned. Past epidemiological evidence did not very often fit the facts.

As noted above, the very recent literature seriously challenges the value of the coliform test as an indicator of hazard to water recreationists. The studies of Moore (1959) and of McCoy (1964) indicate that there is very little hazard from Salmonella when heavily polluted water (by present standards) is used for recreation. Flynn (1965) in Australia flatly states that there is no relationship between coliform count and disease resulting from swimming and feels that the coliform criteria, as presently constituted, do not in any way reflect actual health risks in recreational waters.

The application of the methodology to the health risk associated with coliform concentration reveals the weakness of present methods of criteria setting and at the same time offers a way of establishing a meaningful coliform index. By basing the correlation of the Salmonella and coliform factors as their frequency distribution in a given water, a true picture of the indicator role of E. coli can be derived. This could then be superimposed on the risk curve of the factors of interest, virus and Salmonella, and realistic coliform criteria that are directly related to the level of risk then derived.

It is interesting to note from the examples given that it is the risk of virus infection that will pertain in the establishment of "safe" coliform levels. In contradiction to traditionally held views, the probability of a virus infection increases more rapidly than does the risk from Salmonella.

Fecal Coliforms

Unfortunately, some strains included in the total coliform group have a wide distribution in the environment but are not common in fecal material. To further complicate the problem, some coliforms surviving sewage chlorination may increase exponentially within one or two days' travel downstream (Evans, et al, 1968). This phenomenon, known as

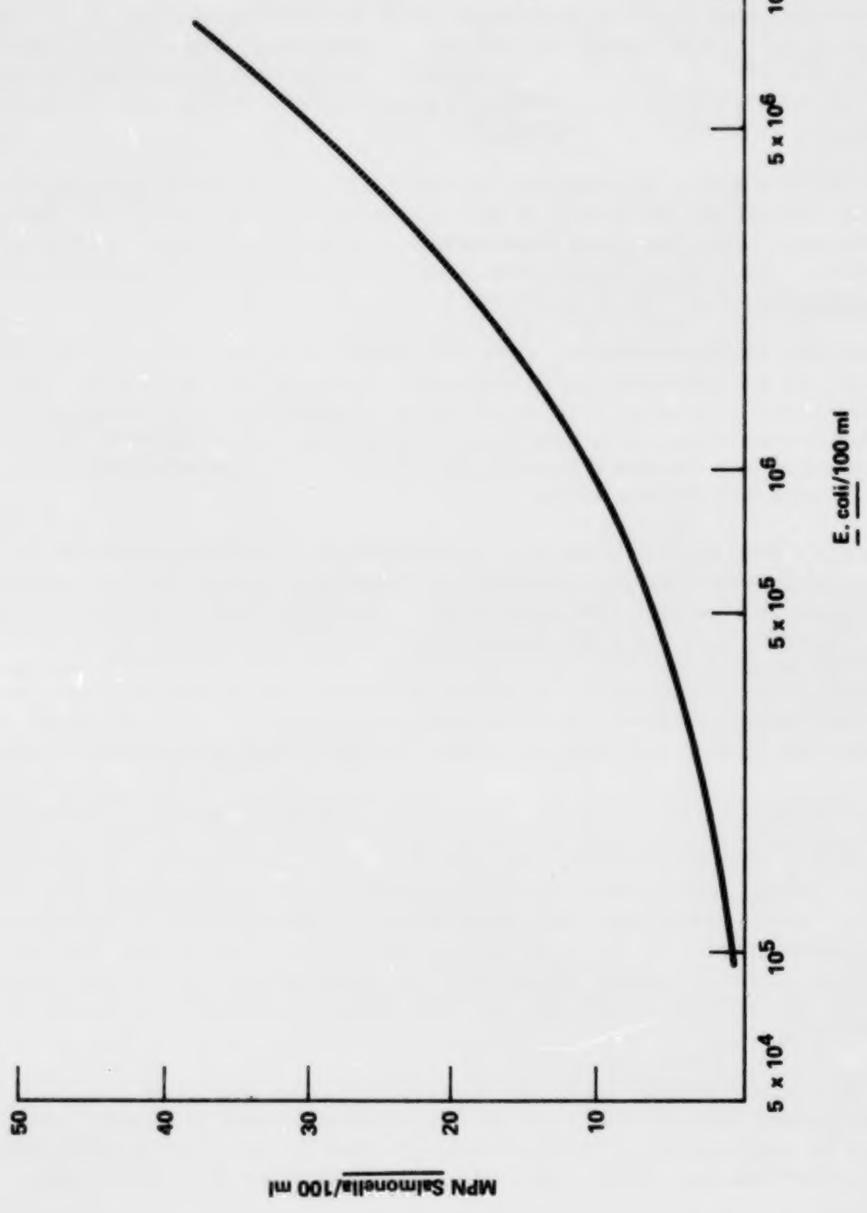


Figure 19. Expanded Portion of Figure 18 Curve

aftergrowth, is associated with the Aerobacter aerogenes portion of the total coliform group. This organism can grow with very minimal nutrients and does not require the complex amino acids or other additives that are necessary for E. coli and other fecal coliform strains. Thus, A. aerogenes is the most responsive coliform bacteria to the stimulation by available nutrients and the associated factors of favorable temperature, pH, and chelation of toxic metal ions by various colloids present in the polluted water.

Other indicator systems have been proposed from time to time, including certain pathogenic bacteria, anaerobic spore-formers, and total bacteria population. For a variety of reasons, these indicator systems have not been found to be satisfactory. However, recent investigation into the fecal coliform sub-group of the total coliform bacteria has shown promise for improving the bacteriological tools used to detect evidence of fecal pollution (Geldreich, 1966).

By definition, the fecal coliform group is composed of organisms that ferment lactose and produce gas within 24 hours at 44.5°C and is more inclusive than the ++--IMViC strain, E. coli, type I. The detection of fecal coliform bacteria by confirmation of all positive presumptive tubes in EC medium at 44.5°C, or by the membrane filter procedure with M-FC (Geldreich, et al, 1965) at 44.5°C is known as the elevated temperature test. In the evaluation of results, all coliforms from the feces of warm-blooded animals are considered fecal coliform strains.

In studies on the occurrence of fecal coliform in the environment, the elevated temperature procedure was shown to have had a 96.3% correlation with coliforms from such fecal sources as humans, cows, pigs, sheep, chickens, turkeys, and ducks by Geldreich and collaborators (1962). Research on the occurrence in the environment of fecal coliform bacteria from dogs, cats, and various rodents, including rabbits, chipmunks, and mice, indicated a 94.5% correlation of the test with fecal origin of these coliform organisms (Geldreich, 1965). This type of pollution could be a major source of the fecal organisms found in residential storm-water runoff and in bathing beach water.

If one assumes that fecal coliforms make up about 15% of the total coliform count (Spino, 1968), then an analysis similar to that of Streeter can be made for the fecal coliforms. In Streeter's work he proposed a Salmonella/coliform ratio of 1/170,000; 15% of this figure would give a Salmonella/fecal coliform ratio of 1/25,000. Using this ratio as a basis, and calculating the risk associated with typical state standards of 1000/100 ml for fecal coliform, a risk of infection of 1/150 is predicted. Obviously this high level of risk would be intolerable; it is, however, incompatible with epidemiological evidence as to disease incidence.

This type of evidence sheds doubt on the validity of fecal coliform standards with respect to assessing the risk of infection with enteric disease. From a review of the published literature, it is apparent that fecal coliform standards are correlated with total coliform incidence, and not to Salmonella or disease incidence. By assuming a constant ratio of TC/FC in water, and setting standards accordingly, the usefulness of fecal coliforms as an indicator is jeopardized. What is urgently needed are more quantitative studies on Salmonella/fecal coliform ratios before realistic risk situations can be established.

Strobel (1968) examined the relationship between fecal coliforms and coliforms for several embayments located on Long Island, New York. The data demonstrated that this relationship varies with the source of pollution, level of treatment provided, characteristics of the receiving waters, and precipitation on the watershed. He concluded that the correlations should be specific for each given water of interest. This fits in with one of the main tenets of the methodology developed in this program in that the factor distributions should be established for each water of interest as part of the monitoring program.

As an example of this type of analysis, the data developed by Strobel for Hempstead Bay was used in Figure 11 and Figure 16 to demonstrate the use of fecal coliforms for establishing risk criteria. The superimposing of the various factor concentrations on the abscissa of the risk curve permits the correlation of new or complementary standards with traditional criteria.

SUMMARY

The traditional concept of using levels of total coliform densities to describe bacteriological acceptability of water bodies has been questioned by many because the presence of coliforms may result from causes other than fecal pollution. Because fecal coliform densities result from pollution by man and other warm-blooded animals, they are more directly indicative of the probable presence of the associated enteric pathogens. However, summarized data from several stream surveys reported over the past few years show little apparent correlation between quantities of total or fecal coliform and the probable isolation of such pathogens as Salmonella and viruses. Salmonellae were isolated by Spino (1966) at total coliform densities of less than 1000/100 ml and fecal coliform densities of less than 150/100 ml. The total coliform density of 1000/100 ml has traditionally been acceptable for recreational waters in many states.

The analysis of available data on Salmonella and virus infectivity along with their relationship to the indicator organisms strongly suggests that present coliform criteria are very conservative. The setting of unnecessarily stringent standards makes the chance of acquiring a disease so small that when an infection does occur it is merely a chance event. This would account for the wide data scatter and apparent unreliability of disease-organism concentration relationships. Once the proper criteria are established, the coliform analysis will in all probability develop into a reasonable predictor of health risk.

SECTION IX

PESTICIDES AS A FACTOR OF RECREATIONAL WATER QUALITY

In presenting the following account of representative information on pesticide/health/environment relationships, the objective has been to develop the thesis that the recreationist enters contaminated water with an acknowledged body burden of chlorinated hydrocarbon pesticides, and that the principal risk he faces may be the raising of his tissue and blood levels to a degree that ultimately results in a clinical expression of a toxic effect. Human contact with the environment represents a spectrum of both intensity and duration of exposure to pesticides, with the general public on the lower end of the scale. People ingest and absorb small amounts of pesticides as residues in foods, and receive further exposure to pesticides applied in their homes and gardens for pest control purposes.

The degree of additional health hazard that is posed by the ingestion of minimal amounts of pesticide-contaminated water by the aquatic recreationist is a matter that awaits resolution. Although no direct evidence in the literature of acute poisoning or chronic-ill effect attributable to primary contact recreation could be found, considerable data are available concerning the relationships between human health and pesticides in the general environment.

Figure 20 illustrates various parameters that should be considered when assessing the hazard faced by the aquatic recreationist as a consequence of exposure to pesticide-polluted water. These parameters include:

(1) The concentration of pesticides in the general environment, which serves as the potential source of contamination of water.

(2) Routes of entry into the water environment.

(3) Known or suspected concentrations of pesticides in water.

(4) Persistence and/or degradation of the pesticide in the water environment.

(5) Potential physiological hazards to man.

The following account represents a sampling of available information on (1) the acute and chronic toxicity of pesticides to man, (2) the amount of pesticides in the diet, (3) the pesticide concentration in surface water, and (4) the epidemiology of pesticide exposure.

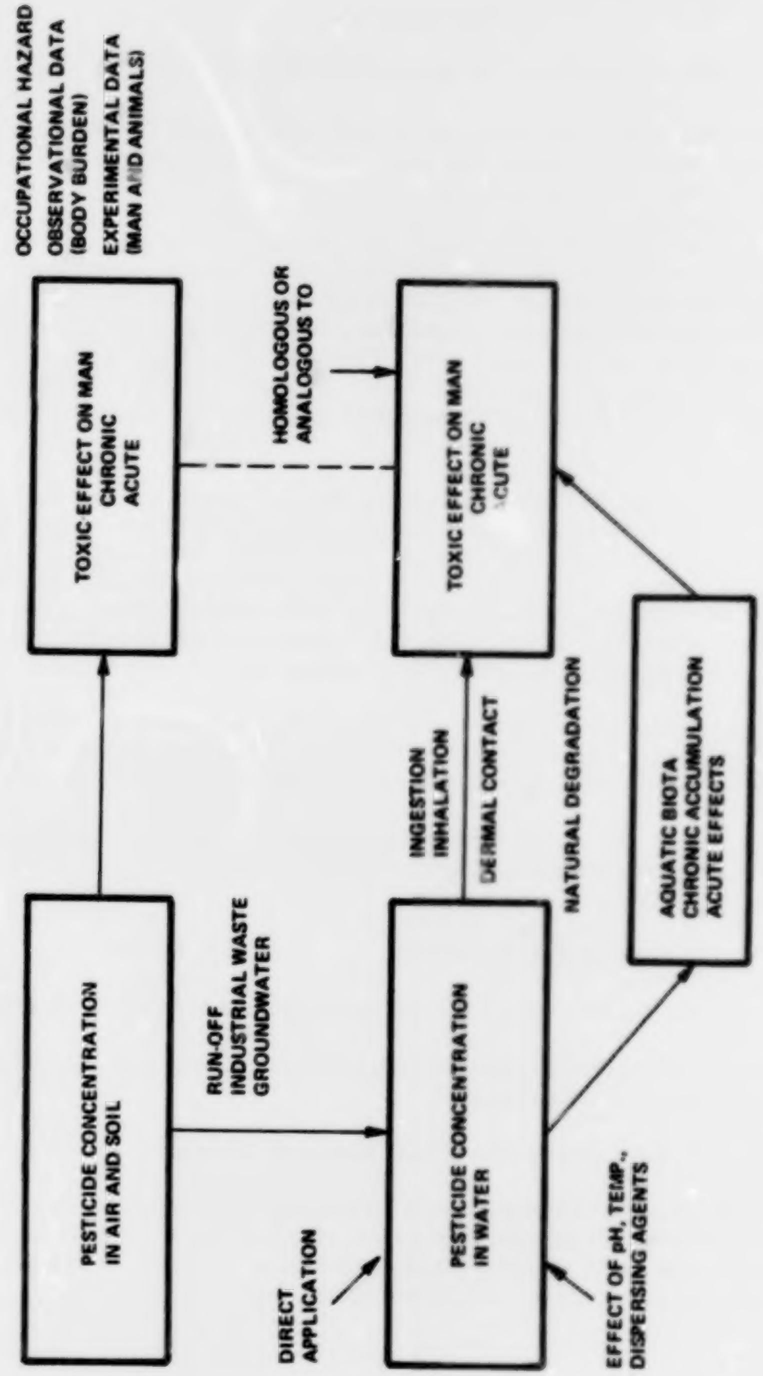

PESTICIDE CONCENTRATION IN AIR AND SOIL
TOXIC EFFECT ON MAN CHRONIC ACUTE
OCCUPATIONAL HAZARD
OBSERVATIONAL DATA (BODY BURDEN)
EXPERIMENTAL DATA (MAN AND ANIMALS)
RUN-OFF
INDUSTRIAL WASTE
GROUNDWATER
HOMOLOGOUS OR ANALOGOUS TO
DIRECT APPLICATION
PESTICIDE CONCENTRATION IN WATER
INGESTION
INHALATION
DERMAL CONTACT
TOXIC EFFECT ON MAN CHRONIC ACUTE
NATURAL DEGRADATION
EFFECT OF pH, TEMP., DISPERSING AGENTS
AQUATIC BIOTA
CHRONIC ACCUMULATION
ACUTE EFFECTS

Figure 20. Various Parameters of the Pesticide-Water-Health Hazard Relationship

ORGANIC PHOSPHORUS INSECTICIDES

The organic phosphorus insecticides (parathion, Chlorthion, demeton, diazinon, Dipterex, malathion, tetraethylpyrophosphate (TEPP)) are among the most toxic to man of the commonly used agricultural chemicals. The organic phosphorus insecticides are all cholinesterase inhibitors. Parathion is one of the most dangerous to humans. Between 1947, when parathion was first introduced, and 1959, there were 100 parathion deaths in the United States. Japan has averaged 336 parathion deaths annually for several years and 100 deaths due to this insecticide occurred in India in 1958. In the same year, there were 67 deaths in Syria and 20 in Jordan (Hayes, 1960). It should be pointed out that malathion is the least toxic of the organic phosphorus group.

Ordinarily there is little, if any, risk from residual concentrations of the organic phosphorus insecticides if they are used properly. Quinby and Lemon (1958) have reported mild parathion poisoning in over 70 workers, however, who handled recently sprayed fruit or other crops. Absorption in these cases was dermal, which probably accounted for the mildness of the reaction.

The toxicity of the organic phosphorus insecticides is directly related to the amount the normal cholinesterase level is lowered in the body. Generally, outward symptoms do not appear until the serum cholinesterase level is lowered by about 30% of normal. This, of course, varies with individuals. Persons chronically exposed to organic phosphorus compounds may reach an equilibrium situation wherein their cholinesterase level remains at some lower level. An accidental added exposure may then result in much more severe effects than a normal individual would experience at the same dose level.

Although numerous human volunteer studies have been carried out with the various pesticides, most such work has been conducted at conservatively low exposure levels to avoid permanent damage to the subjects. This type of testing is suitable for establishing safe levels, but does not provide information on dangerous limits. Dependence for this latter type of information is thus placed on cases of accidental poisonings or suicides. In both cases, it is rarely possible to compute the ingested doses with any accuracy.

Several cases of poisoning have occurred that did allow estimation of the dosages ingested. These reports, along with reported exposures of volunteers, are summarized in Table 30. A plus-minus system is used to record effects since in many cases different types of clinical evaluations were used (serum cholinesterase, urine, or the clinicians description of the symptoms). In many cases, only the dosage ingested was reported and the weights of the subjects were not reported. This data was divided by 70 to derive a mg/kg dosage for an "average" man.

TABLE 30

Organic Phosphorus - Pesticides

Dosage mg/kg	% Decrease	Effect on Humans *	Material	Reference
1.6	-	▭	parathion	Goldblatt 1950
0.73-1.4	-	▭	parathion	Seifert 1954
1.3	-	▭	TEPP	Grob & Harvey 1949
0.84	-	-	malathion	Mattson & Sedlar 1962
0.34	-	+++	TEPP	Grob & Harvey 1949
0.34	25	++	malathion	Moeller & Rider 1962
0.32	-	+	malathion	Moeller & Rider 1962
0.21	-	-	malathion	Moeller & Rider 1962
0.16	-	-	malathion	Mattson & Sedlar 1960
0.15	-	±	Delnav	Weir & Kelber 1962
0.12	-	+	EPN	Moeller & Rider 1962
0.10	-	▭	parathion	Kagaratnam et al 1960
0.10	33	+++	parathion	Edson et al 1964
0.10	-	±	dioxathion	Trawley et al 1963
0.10	15	+	methyl parathion	Moeller & Rider 1962
0.097	33	++	parathion	Edson 1957
0.081	-	-	EPN	Moeller & Rider 1962
0.081	10-15	+	parathion	Moeller & Rider 1962
0.075	-	-	Delnav	Weir & Keller 1962
0.065	-	±	parathion	Edson 1957
0.061	-	-	parathion	Moeller & Rider 1961
0.060	77	+++	schraden	Edson et al 1964
0.056	77	+++	schraden	Edson et al 1964
0.05	-	-	parathion	Rider 1958
0.05	15	+	demeton	Moeller & Rider 1962
0.05	-	-	dioxathion	Trawley et al 1963
0.04	-	-	parathion	Moeller & Rider 1961
0.032	-	-	parathion	Edson 1957
0.025	-	-	parathion	Rider 1958
0.016	-	-	parathion	Edson 1957
0.014	25	+	schraden	Edson et al 1964
0.0034	25	+	dimefox	Edson et al 1964
0.0020	-	-	dimefox	Edson et al 1964

* Legend:

± = baseline change
+ = 1-25%
++ = 25-50%
+++ = over 50%
▭ = death

} % reduction in cholinesterase

In spite of these drawbacks, Table 30 serves a useful purpose by showing a graded response from the high dosages of approximately 1 mg/kg, which is always fatal, down to relatively safe dosages below 0.05 mg/kg. Malathion shows up as one of the safer organic phosphorus insecticides while schraden is seen to be extremely toxic. The other interesting features of this table is the rather narrow range between no effects and extreme toxicity (e.g., 0.03 mg/kg (safe) - 0.1 mg/kg (death) for parathion) for these compounds. This may require a more liberal than usual safety factor since any appearance of symptoms is a dangerous situation.

Although the organic phosphorus insecticides exhibit their toxic effects over a rather narrow range, there is a wide difference in the toxicity between different compounds. This is further complicated, from the viewpoint of RWQ criteria, by the broad spectrum of solubilities - from the fully water miscible systemics, schraden and dimefox, to the almost insoluble parathion, TEPP, etc. Fortunately, however, most organic phosphorus insecticides have a relatively short life time in a water environment, being quickly hydrolyzed to innocuous materials. As will be pointed out later, these pesticides would cause mass fish kills in a water long before human toxic levels can occur. This would serve as a strong warning to humans to stay out of such water.

CHLORINATED HYDROCARBON INSECTICIDES

Insecticides in the chlorinated hydrocarbor group include DDT, BHC (and lindane), chlordane, chlorobenzilate, TDE (DDD), Dilan, aldrin, dieldrin, endrin, heptachlor, and its epoxide, toxaphene, and methoxychlor. Two notable members of this group are DDT and BHC. Fortunately, these two insecticides have produced no authenic cases of poisonings (Barnes, 1958; Hayes, 1960) from ingestion of treated foods. However, there have occurred several cases of accidental poisonings and attempted suicides. The problem of accurately assessing the dosage ingested is very difficult in most of these cases.

Hsieh (1954) reported an incidence of accidental poisoning from DDT, involving eleven persons who had eaten contaminated pork dumplings. This study is noteworthy in that the author went into an elaborate process to determine the amount of DDT ingested by each individual. This was reported along with the severity of clinical symptoms.

Studies with volunteers have generally been carried out at relatively low dosages. The effect of exposure has shown considerable individual variation with a graded response increasing in severity as dosage is increased. The volunteer studies and cases of accidental poisonings or suicides where the dosage ingested can be calculated are presented in Table 31. Three deaths were reported but in each case there appears to be confusion as to whether the death was pesticide induced or caused by pulmonary edema resulting from the solvent used to dissolve the pesticide. A plus-minus scale is used to rank severity of intoxication since the

TABLE 31

Toxicity to Humans of Chlorinated Pesticides

Pesticide Concentration			
Pesticide	mg/kg	Severity	
DDT	500.0	■	Baden-Steel - see Stammers (1947)
DDT	285.0	****	Hayes - 1954
BHC	180.0	■	Kwocjek 1950 - in Durham
DDT	120.5	****	Hsieh 1954
DDT	105.0	■	Hill & Robinson - Stammers (1947)
DDT	41.9	***	Hsieh 1954
DDT	37.2	***	Hsieh 1954
DDT	28.9	***	Hsieh 1954
DDT	20.0	***	Velbinger
DDT	20.0	***	Hayes 1955
DDT	18.4	**	Hsieh 1954
DDT	17.5	**	Hsieh 1954
DDT	16.3	*	Hsieh 1954
DDT	16.0	***	Hayes 1959
DDT	13.5	*	Velbinger
DDT	11.1	0	Neal, Sweeney, etc. 1946
DDT	10.3	0	Neal, Sweeney, etc. 1946
DDT	10.0	*	Hayes 1955
DDT	9.5	**	Velbinger
DDT	7.1	0	Neal 1946
DDT	6.7	0	Hsieh 1954
DDT	6.7	0	Velbinger
DDT	6.0	*	Hsieh 1954
DDT	6.0	*	Hayes 1959
DDT	5.1	0	Hsieh 1954
methoxychlor	3.38	=	Hayes
methoxychlor	3.37	0	Domenjers
methoxychlor	2.0	0	Stein 1965
methoxychlor	1.0	0	Stein 1965
methoxychlor	0.5	0	Stein 1965
methoxychlor	0.5	0	Hayes 1956
dieldrin	0.21	0	Hunter 1967
dieldrin	0.05	0	Hayes
dieldrin	0.01	0	Hunter 1967

Legend: ■ = death

**** = cysnosis vomiting

*** = heart convulsions

** = tremor, convulsions, malaise

* = headache

0 = no reaction

description of symptoms is in part a subjective judgment of the attending physician.

Although DDT is stored in body fat and the effects are therefore cumulative, no case of chronic poisoning analogous to that produced by arsenic, lead, or mercury have been described for DDT or related insecticides (Hayes, 1960). However, there have been a few cases reported in the literature of agricultural poisonings with dieldrin, aldrin, and endrin. Dermal exposure to dieldrin has led to epileptoid convulsions in man, although the effects have generally been transitory.

Because of the low water solubility of most of the chlorinated hydrocarbon they cannot be considered an important source of hazard to water recreationists. It would be almost impossible for a recreationist to ingest the smallest dose (6.0 mg DDT/kg) shown on Table 31 as producing an effect. If an individual is to ingest 6.0 mg DDT/kg, a 70 kg man would require a total intake of 420 mg. This in turn would have to be contained in the 10 ml assumed to be normally ingested during recreational activities. The required concentration in the water would have to be 42 g/l. This if far above the solubility limit of about 1-10 μg/l for DDT.

These levels are, of course, far above any concentrations monitored in natural waters. Because this factor was not considered of importance to recreational waters, a convolution was not carried out.

CHRONIC TOXICITY OF PESTICIDES

Two types of injury must be considered in assessing the health hazards of pesticide residues. The first is the possibility of an acute illness resulting from residues ingested on a single day or in a few days. The second is the long-term effects that may accrue after ingesting small quantities of residues daily for many years. On the basis of documented knowledge of the effect of various pesticides on experimental animals and on man, and of the levels of the chemicals present as residues on food products, illness from short-term exposure would not be expected to occur. This conclusion is supported by epidemiologic evidence. There have been no known cases of illness in the United States from insecticide residues on foods when formulations have been used according to directions (Hayes 1960). However, there have been several instances in which insecticides used improperly on foods have led to acute poisoning soon after ingestion. Improper use may include excess application of the insecticides, and/or inadequate treatment of the produce to remove the insecticides prior to marketing (Durham, 1963).

PESTICIDES IN THE DIET

Of great importance for public health is a knowledge of the amount of pesticide residues on foods and how much is actually consumed by the general population. Indeed, pesticides ingested with food provides a background level of intake that may mask the added exposure to the population from water related recreational activities.

The DDT and DDE content of a group of complete, prepared meals from restaurants and institutions were determined by Walker and his co-workers (1954). A total of 179 individual portions of food, representing 86 different items, was included in the 18 restaurant and 7 institutional meals tested. Fifty food portions contained no detectable DDT. However, DDT was found to be present in detectable but very small quantities in all meals tested. Generally, those foodstuffs cooked in fat, and those containing meat or butter were found to have a higher DDT content than other foodstuffs. The DDE content of the various foods tested was roughly proportional to the DDT content. If, in one day, an individual had consumed the three meals that contained the largest amounts of DDT, his total DDT intake would have been 0.388 mg. The average DDT intake, based on all meals tested, was 0.184 mg/day. This amount is equivalent to a DDT dosage of about 0.002 mg/kg - day for a man of average size (70 kg) or to a DDT concentration of about 0.31 mg/kg in the total dry diet. It was felt that no meal tested contained enough DDT to be considered a toxicological hazard on the basis of the estimated chronic oral toxicity of the compound.

Hayes, et al (1956) determined the DDT and DDE content of 16 meals and found the average DDT intake to be 0.202 mg, as compared with an average daily DDE intake of 0.050 mg. Other studies by Hayes (1958) and Durham (1961) revealed similar results.

Figure 21 and Table 32 (extracted from Duggan and Lipscomb, 1969) are presented to illustrate the average incidence of pesticide residues in representative food samples and to indicate the calculated daily intake of a 70 kg man. Table 33 was prepared by the above authors to compare these values with the acceptable daily intake proposed for some significant pesticide chemicals by the Food and Agricultural Organization of the United Nations and the World Health Organization Export Committee on Pesticide Residues. Acceptable daily intake is defined as "the daily dosage of a chemical which, during an entire lifetime, appears to be without appreciable risk on the basis of all the facts known at the time. 'Without appreciable risk' is taken to mean the practical certainty that injury will not result even after a lifetime of exposure."

Examination of the tables reveals that no acceptable daily intake value was exceeded during the four years of the referenced study, and the calculated daily dietary intake for practically all pesticide chemicals is one order of magnitude or more below that considered safe by the FAO/WHO Expert Committee. The average daily intake of all chlorinated organic pesticide residues was 0.0013 mg/kg of body weight. The average dietary intake for all organic phosphorus compounds was 0.0001 mg/kg of body weight.

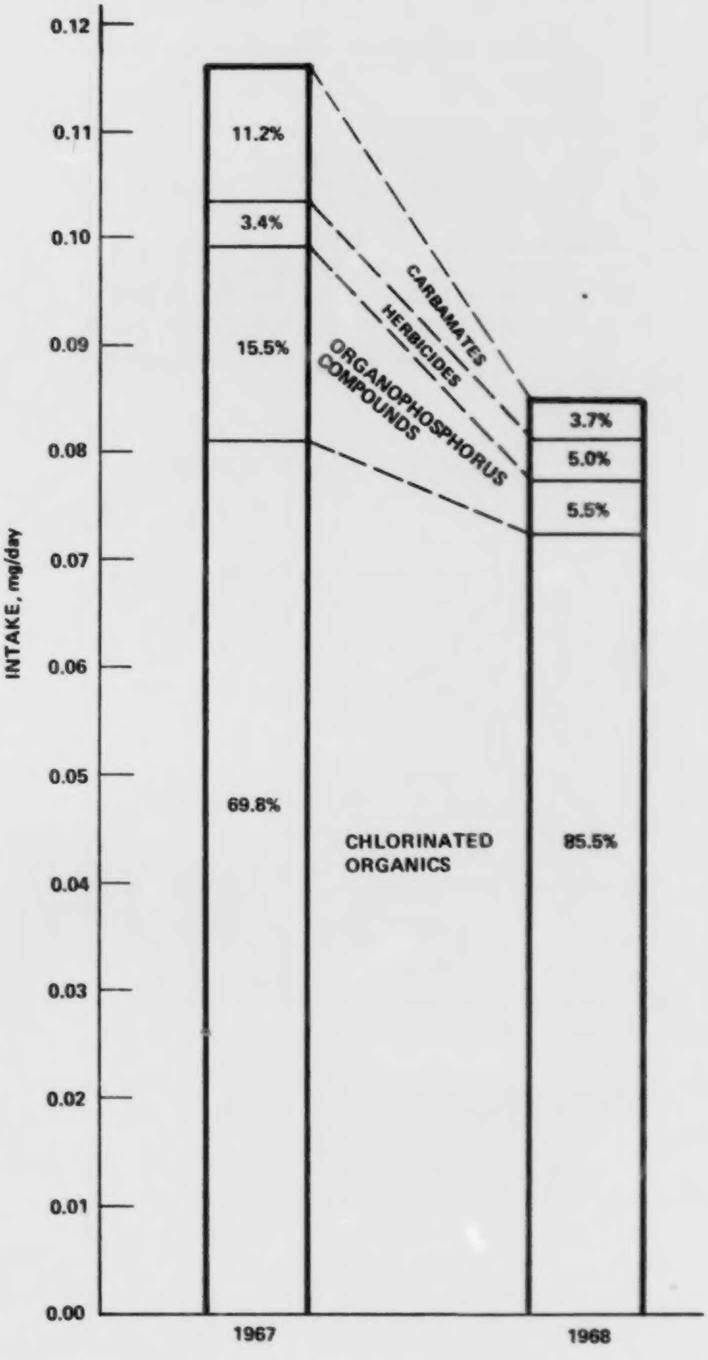


Figure 21. Distribution of Pesticide Residues by Chemical Class, 1967-68

TABLE 32

Average Incidence and Daily Intake of 15 Pesticide Chemicals
(After Duggan and Lipscomb, 1969)

	1965		1966		1967		1968	
	Percent Positive Composites*	Daily Intake, mg	Percent Positive Composites**	Daily Intake, mg	Percent Positive Composites***	Daily Intake, mg	Percent Positive Composites***	Daily Intake, mg
DDT	17.5	0.031	37.3	0.041	38.6	0.026	49.2	0.019
DDE	31.5	0.018	33.0	0.028	31.1	0.017	37.5	0.015
TDE	19.4	0.013	25.7	0.018	28.9	0.013	31.1	0.011
Dieldrin	18.5	0.005	21.3	0.007	15.3	0.004	15.6	0.004
Lindane	15.8	0.004	12.3	0.004	10.6	0.005	15.3	0.003
Heptachlor epoxide	13.4	0.002	12.0	0.003	8.9	0.001	13.1	0.002
BHC	6.5	0.002	6.0	0.004	8.9	0.002	9.7	0.003
Malathion	-	-	5.3	0.009	3.6	0.010	1.9	0.003
Carbaryl	7.4	0.15	2.7	0.026	1.1	0.007	-	-
Aldrin	5.6	0.001	3.7	0.002	3.3	0.001	3.9	0.001
2, 4-D	4.2	0.005	3.0	0.002	1.7	0.001	0.6	0.001
Diazinon	-	-	3.0	0.001	0.3	0.001	0.3	0.001
Kelthane	0.5	0.003	3.7	0.002	5.6	0.012	4.7	0.010
PCB	1.4	0.001	3.3	0.006	2.2	0.001	1.9	0.001
Endrin	2.8	0.001	2.0	0.001	1.7	0.001	1.1	0.001

* 216 composites examined
** 312 composites examined
*** 360 composites examined

NOTE: Sampling was done bimonthly beginning in June and ended in April of the years shown.

TABLE 33

Dietary Intake of Pesticide Chemicals
(After Duggan and Lipscomb, 1969)

Compound	FAO-WHO Acceptable Daily Intake	Daily Intake, mg/kg Body Weight			
		1965	1966	1967	1968
Aldrin	-	0.00001	0.00004	0.00001	0.00001
Dieldrin	-	0.00008	0.00009	0.00005	0.00005
Total	0.0001	0.00009	0.00013	0.00006	0.00006
Carbaryl	0.02	0.0021	0.0005	0.0001	-
DDT	-	0.0004	0.0005	0.0004	0.0003
DDE	-	0.0003	0.0003	0.0002	0.0002
TDE	-	0.0002	0.0002	0.0002	0.0002
Total	0.01	0.0009	0.0010	0.0008	0.0007
Gamma BHC (Lindane)	0.0125	0.00007	0.00006	0.00007	0.00004
Bromide *	1.0	0.39	0.22	0.29	0.41
Heptachlor	-	0.000003	-	0.000001	0.000001
Heptachlor epoxide	-	0.00003	0.00005	0.00002	0.00003
Total	0.0005	0.000033	0.00005	0.000021	0.000031
Malathion	0.02	-	0.0001	0.0002	0.00004
Parathion	0.005	-	0.00001	0.00001	0.000001
Diazinon	0.002	-	0.00002	0.000001	0.000001
BHC	-	0.00003	0.00004	0.00003	0.00004
Kelthane	-	0.00004	0.00015	0.00018	0.0001
Endrin	-	0.000009	0.000004	0.000004	0.00001
All chlorinated organics	-	0.0012	0.0016	0.0012	0.0011
All organophosphates	-	-	0.00014	0.00025	0.00007
All herbicides	-	0.00012	0.00022	0.00005	0.00006

*Total bromides present include naturally occurring bromides.

NOTE: Sampling was done bimonthly beginning in June and ended in April of the years shown.

PESTICIDES IN WATER

Pesticides may enter the water supply by direct, intentional application; by inadvertent drift into water from adjacent spraying operation; or, perhaps more commonly, by leaching of pesticide-treated areas within a watershed (Durham, 1963).

Fortunately, the great susceptibility of fish to many of these insecticides gives an easy clue to significant contamination of streams. For example, endrin is toxic to certain species of fish at concentrations of less than 1 ppb. The toxicity of toxaphene to fish is of the same order of magnitude as that of endrin. The presence of many other chlorinated hydrocarbon pesticides at concentrations as low as a few parts per billion may be detected by their effect on fish. The carbamate insecticide, Sevin, and the organic phosphorus compound, Guthion, also fall within this range of toxicity to fish. The latter group of insecticides is not very important as water pollutants, however, due to their relatively rapid hydrolysis rates (Johnson, 1968).

The U.S. Public Health Service instituted a nation-wide survey of fish kills as a means of detecting water pollution, utilizing the high toxicity of the newer synthetic pesticides to fish as the indicator of insecticide content. In the first four months of the survey (June - September 1960), more than 200 individual reports of fish kills were received. In 76 cases (38 percent), agricultural chemicals were implicated as the etiological agent (Cottam, 1960).

Nicholson (1960) made a very thorough study of the pesticide content of a river system that drains a 400 square-mile cotton-farming area of Alabama in which pesticides, particularly toxaphene, DDT, and lindane, were intensively used. Toxaphene (maximum level 0.0004 mg/l) and lindane (maximum level 0.00075 mg/l) were detected in the stream. DDT was not detected. The level of pesticides in the water was not decreased by the municipal water-treatment process. Toxaphene was found in the water all year long, but heaviest concentrations coincided with application to fields. The contamination was general throughout the watershed. The insecticides apparently ran off the land surface and dissolved in the water. No effects of these pesticides on the aquatic life of the stream were noted.

The U.S. Geological Survey, in cooperation with the EPA, is currently operating a pesticides monitoring network, which is part of a program for continuous surveillance of pesticides in surface waters at selected sites throughout the U.S. Manigold and Schultz (1969) reported on the most recent findings in certain selected western streams. They presented data from a network of 20 sampling stations. Compounds determined included the chlorinated insecticides aldrin, DDD, DDE, DDT, dieldrin, endrin, heptachlor, heptachlor epoxide, and lindane. Also studied were the chlorinated herbicides, 2,4-D, 2,4,5-T and silvex.

All of these were detected at one time or another. DDT was the most frequently occurring insecticide, and 2, 4-D was the most commonly found herbicide. The amounts observed were small; the maximum concentrations of DDT and 2, 4-D were 0.12 and 0.35μg/liter, respectively. Concentrations were highest in the water samples that contained appreciable amounts of sediment.

Other evidence presently available would indicate that pesticide residues may be present, although at extremely low levels (in the range of 0.001 mg/l) in streams draining watersheds that have been treated with chlorinated hydrocarbon pesticides. At a concentration level of 0.001 mg/l, an individual's average daily intake of two liters of water would represent only about 0.002 mg of residual insecticide. According to present knowledge, the amount of even the most toxic pesticide would be significantly below the level that is hazardous on an acute or subacute basis (Durham, 1963).

The carbon filter-chloroform extract technique has often been used to monitor selected waters for various organic chemical groups. Although the method does not identify specific pesticides, it does quantify various classes of compounds that include most of the pesticides. This method gives an analysis of the total contamination of the water by different chemical groups and permits the establishment of general criteria instead of a series of individual pesticide values.

An example of such a program is the National Water Quality Network of the Public Health Service. The Ohio River from Huntington, West Virginia, to Cairo, Illinois, was selected for analysis, with October 1957 to September 1958 being the time span covered. Most of the pesticides of concern would appear in the chloroform extracts from the carbon columns, the chlorinated hydrocarbons in the aromatic fraction and the organic phosphorus compounds in the oxygenated-compounds fraction.

Figure 22 presents the distribution of total chloroform extractables in the river. A histogram of the data was prepared, and points selected randomly from the histogram were used as inputs into the BSTFT program. The Gamma distribution was selected as the most likely representation of the sampled universe (Table 34), although the probabilities are also high that Weibull or lognormal distributions would also be acceptable.

A similar analysis was carried out for the aromatic fraction. Here the 3-parameter Weibull distribution was selected from the data in Table 35 as presented in Figure 23. The highest level of 48μg/l is about 70% of water saturation and occurs at the asymptotic portion of the curve.

The cumulative frequency distribution for oxygenated compounds is

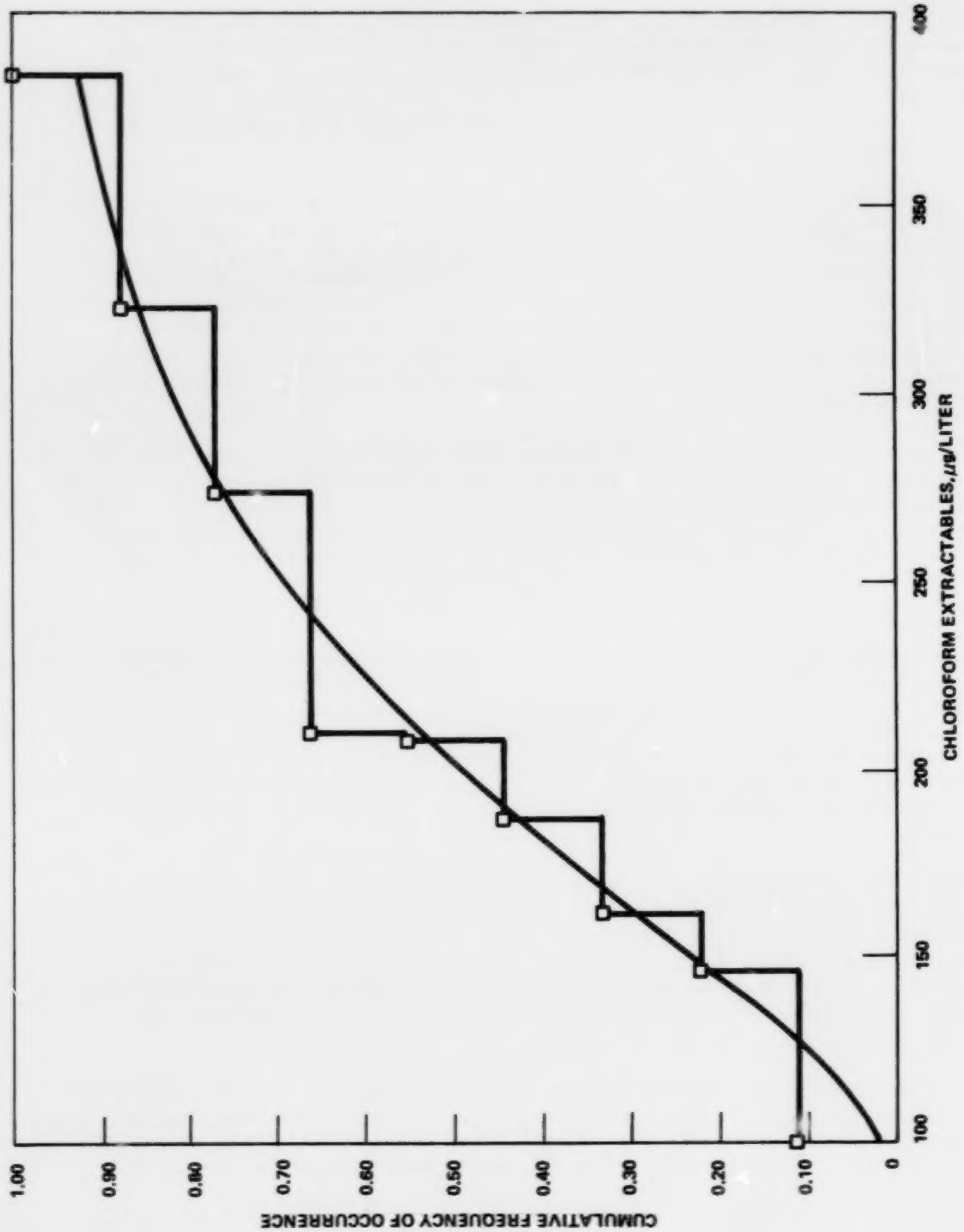


Figure 22. Pollutants in Ohio River Water - Total Chloroform Extractables

TABLE 34

Pollutants in Ohio River Water - Total Chloroform Extractables

Name of Distribution	"d" Statistic	Probability Data Came From Cited Distribution
Normal	0.224	0.755
Lognormal	0.155	0.982
Exponential	0.343	0.240
Gamma	0.127	0.999
Weibull	0.134	0.997

Conclusion: It is not unreasonable to assume that the Gamma distribution ($\hat{c} = 90.167$, $\hat{\beta} = 67.630$, $\hat{\alpha} = 1.9510$) best represents the universe from which the data were sampled

TABLE 35

Pollutants in Ohio River Water - Aromatics (Chlorinated Hydrocarbons)

Name of Distribution	"d" Statistic	Probability Data came from Cited Distribution
Normal	0.327	0.291
Lognormal	0.173	0.951
Exponential	0.248	0.637
Gamma	0.227	0.740
Weibull	0.165	0.967

Conclusion: It is not unreasonable to assume that the Weibull distribution ($\hat{c} = 4.000$, $\hat{\theta} = 8.7775$, $\hat{k} = 8.077 \times 10^{-1}$) best represents the universe from which the data were sampled.

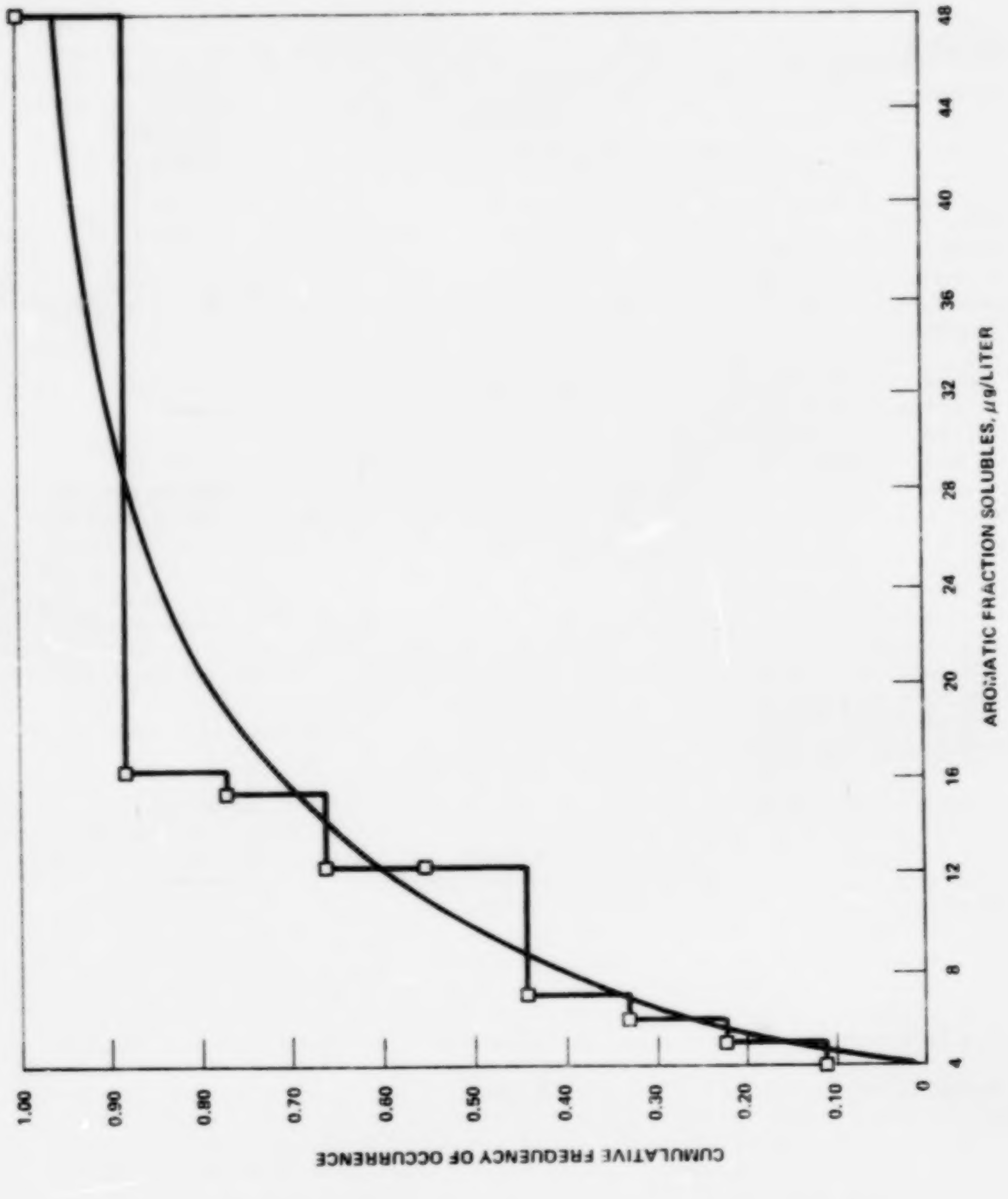


Figure 23. Pollutants in Ohio River Water - Aromatic-Soluble Fraction (Chlorinated Hydrocarbons)

presented as a log normal distribution in Figure 24 based on the analysis in Table 36.

EPIDEMIOLOGY OF PESTICIDE EXPOSURE

A review of the literature indicates that the problem of chronic effects in man of continued ingestion or absorption of minute amounts of chlorinated hydrocarbon pesticides has not been satisfactorily resolved. It is true that for each commerically available pesticide, long-term experiments have been performed in rats, dogs, and other animals. Such experiments in rats have included study of the progeny for three generations. Similar feeding experiments have been carried out in dogs for periods as long as two years. Zapp (1965), however, points out that in spite of the fact that no effects have been seen in the dogs and in three generations of rats with daily doses many times that to which humans are constantly subjected, there is still doubt that the results can be projected to man or be extrapolated to a whole lifetime of exposure in man.

There exists significant data that imply the innocuousness in man of prolonged small doses of chlorinated hydrocarbon pesticides. Hayes, et al (1956) measured the effect of known repeated oral doses of DDT in man, where human volunteers were fed either 3.5 or 35 mg of DDT per day. This was about 20 and 200 times, respectively, as much as Walker, et al (1954) found in the normal diet. No complaints, symptoms, or laboratory findings suggestive of toxic effects of retained pesticides were found in these subjects after 18 months of continuous intake. In another approach, Stein and Hayes (1964) investigated by questionnaire over 2,000 employees who worked in direct contact with pesticides for many years. They found no greater incidence or degree of illness among those involved, than in relatives or other employees who had no special exposure to pesticides. Additional studies of this nature are reviewed by Durham (1963), and more recently by Yeary (1966) (see Figure 25).

Hoffman, et al (1967) engaged in a different attempt at evaluating the potential chronic toxicity of chlorinated pesticides. They determined the extent of storage of these pesticides in the fatty tissues of 994 men and women who died of a variety of diseases, for 688 of whom complete autopsy records were available. An attempt was made to find the degree of correlation between the fat-pesticide levels and the presence or absence of pathological changes in the various organ and tissues. The principal pesticide found was DDT, 72.2% of which was in the form of its more innocuous derivative, DDE. The sum of DDT and DDE averaged 9.6 mg/kg with a standard deviation (SD) of 7 mg/kg.

Hexachlorcyclohexane (BHC, HHC), in the form of its γ-isomer, lindane, or one or more of the other isomers, showed an average concentration in human tissue of 0.48 mg/kg with a standard deviation of 0.54 mg/kg. There were 35 inordinately high values for DDE plus DDT and 11 for hexachlorcyclohexane. These probably represented intakes from sources other than foods. If they were omitted, the mean for DDE plus DDT

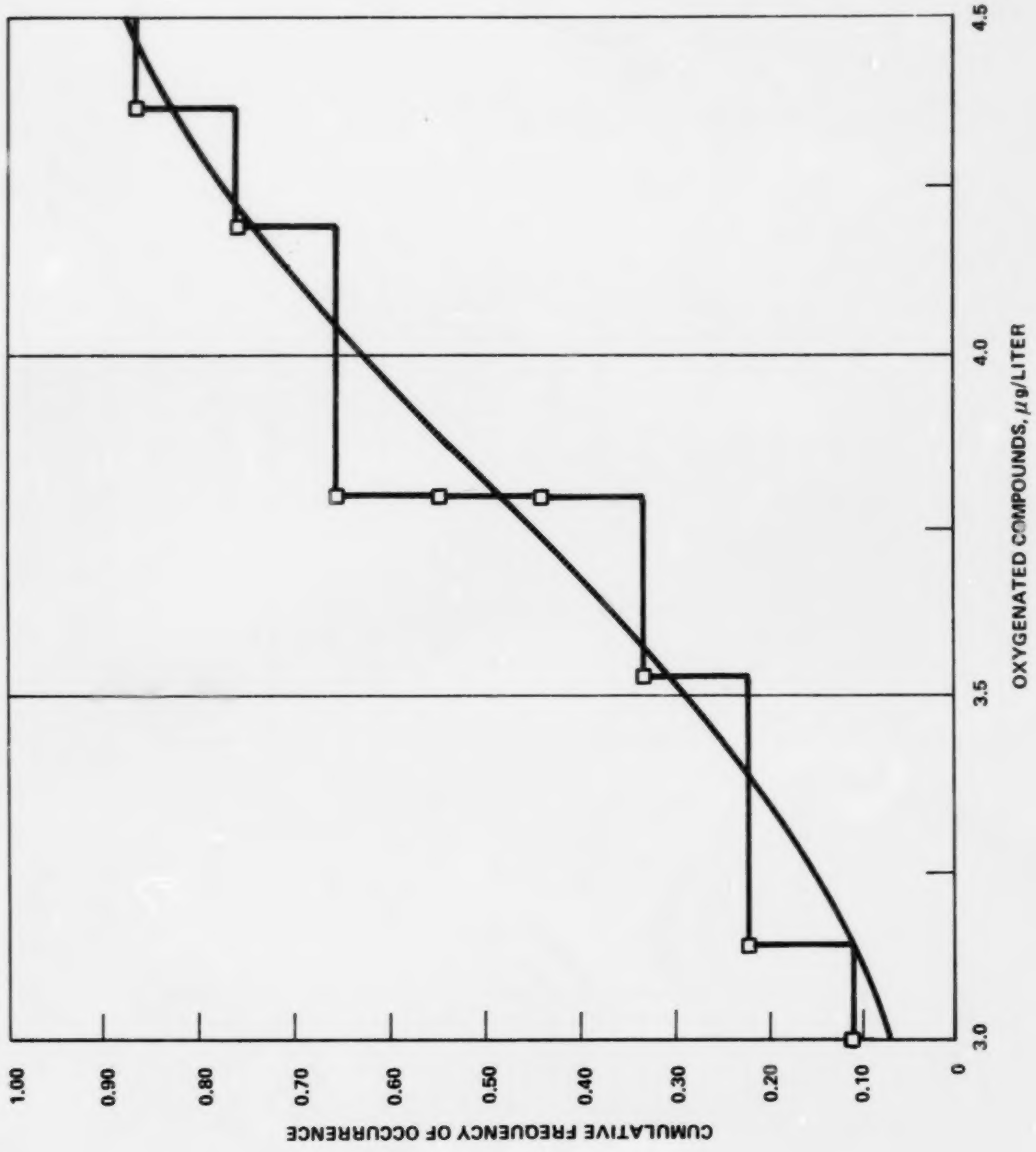


Figure 24. Pollutants in Ohio River Water - Oxygenated Compounds (Phosphates, Esters)

TABLE 36

Pollutants in Ohio River Water -

Oxygenated Compounds (Organophosphorus Compounds, Esters, etc.)

Name of Distribution	"d" Statistic	Probability Data came from Cited Distribution
Normal	0.260	0.577
Lognormal	0.178	0.937
Exponential	0.312	0.344
Gamma	0.218	0.784
Weibull	0.186	0.914

Conclusion: It is not unreasonable to assume that the Lognormal distribution ($\hat{\bar{y}}$ - 3.8431, $\hat{\sigma} y$ = 5.0716×10^{-1}) best represents the universe from which the data were sampled.

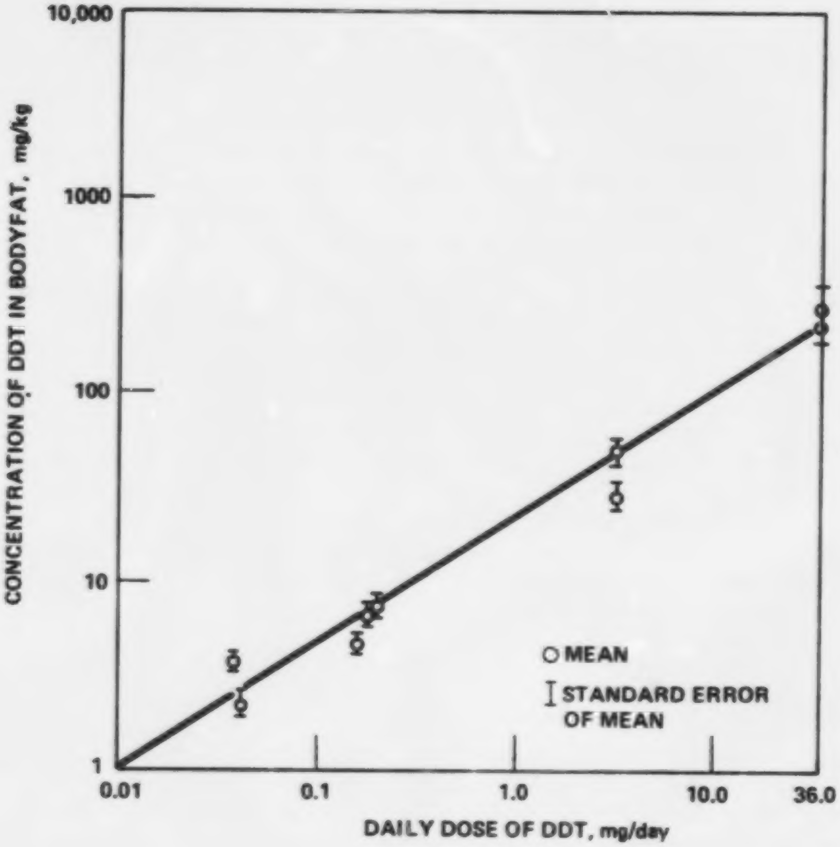


Figure 25. Relationship Between the Concentration of DDT in the Bodyfat of Man and the Daily Dose

would be 8.8 ± 5.5 mg/kg, and for hexachlorcyclohexane 0.44 ± 0.26 mg/kg.

Dieldrin was not present in 103 samples of 221 analyzed for this pesticide. The remaining values ranged from 0.01 to 1.39 mg/kg. The mean for the 221 samples was 0.14 ± 0.20 mg/kg. Heptachlor epoxide was found in 472 of 505 specimens, the mean being 0.16 ± 0.11 mg/kg.

There did not appear to be any evidence of increased body burden of DDT products in the general population since 1951, as compared with previous studies (Quimby, 1965; Dale & Quinby, 1963; Hoffman, et al 1964). The levels for specimens in the above study obtained in 1964 through 1966 were lower than those obtained from persons who died in 1962 and 1963. A statistical survey of the pathological changes in the tissues examined in the 688 autopsies showed that there was no significant correlation between the levels of DDE plus DDT and hexachlorcyclohexane in the fat and presence or absence of abnormalities in these tissues. The authors concluded that their findings were in accord with other evidence that the amounts of DDT products and hexachlorcyclohexane stored in the fat of the general population had not proved harmful.

The effect on animals, including man, of the various pesticides studied seems to follow a normal dosage response curve, meaning that small amounts are probably harmless while larger doses may produce poisoning. Although a number of the common pesticides, including DDT, are stored in the body, this storage is not indefinitely cumulative (Figure 26). With tolerated doses of DDT, man approaches storage equilibrium in about one year, and does not store any more of the compound regardless of how long the dosage is continued.

As already stated, the chronic effects of exposure to chlorinated hydrocarbons are ill-defined. It is doubtful that the very low concentrations found in waters would significantly raise the body burdens. However, instances can occur wherein large amounts of pesticides are released into a water and although below the acute level could bring about transitory increases in body burdens.

There is no information available to indicate how great a body burden an individual can withstand before toxic effects become evident, although doses 7,500 times greater than that to which the general population are exposed have been used. This data would be useful since a series of curves relating dietary intake to equilibrium levels could then be constructed and used to establish the chronic intake level.

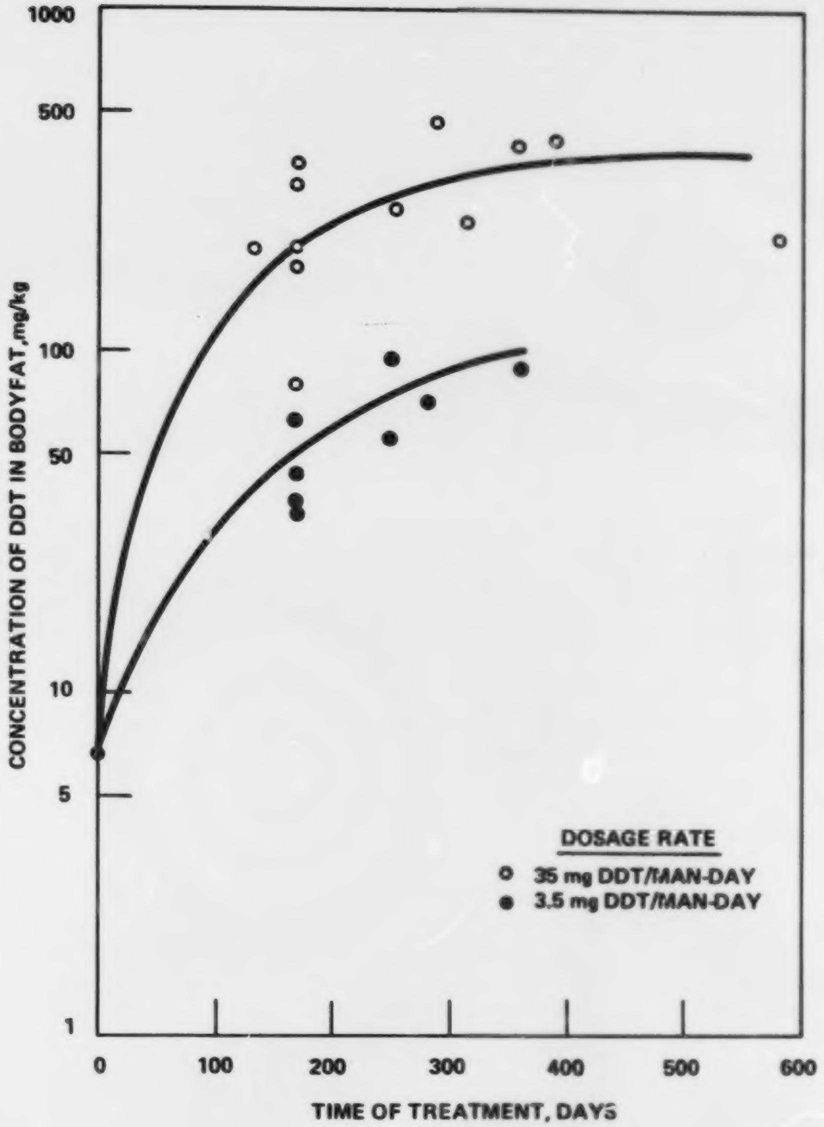


Figure 26. Increase of the Concentration of DDT in the Bodyfat of Men with Continuing Intake

SECTION X

TEMPERATURE AS A FACTOR OF RECREATIONAL WATER QUALITY

Temperature is another factor that is important to the recreationist. Man's survival is endangered at both extremes of the temperature scale. Within a given temperature range, 60 - 85°F, man can function rather comfortable over long periods of time. As the temperature diverges from each end of this range, certain effects are frequently observed. These initially range from purely subjective discomfort to subtle changes in the efficiency of performing simple tasks. As the temperature stress increases, a higher order of psycho-physiological disturbances occur with an increased frequency of errors and further reduced efficiency in task performance. At the furthest temperature extremes, there is a definite loss in work capacity with a physiological strain on the heart and circulatory system and other effects on water and salt balance regulation in the body. These disturbances may culminate in shock and even death.

HIGH TEMPERATURES

Body heat balance is a physiological requirement for comfort and health. Under normal conditions, the rate of heat production (metabolism) is just balanced by the heat loss to the environment. An increase in heat production brought on by exercise must be compensated for by an equal increase in heat loss. In water, this excess heat would normally by carried off by conduction. If the water temperature is above skin temperature, heat that is generated through activity will cause the body temperature to increase. This rise in body temperature can be accommodated by reestablishing a heat balance at the elevated temperature. Although a moderate body temperature rise is acceptable, any substantial increase is accompanied by severe strain (Belding and Hatch, 1956).

Haldane (1905) defined the limit of endurance as the most extreme heat exposure that can be withstood without an abnormal rise in body temperature. Later work demonstrated that the effective temperature for heat tolerance is related to ambient temperature, humidity, and air motion for different levels of activity. By analogy to an unclothed worker in a moisture saturated environment, a swimmer expending about 1200 Btu/hr would have a safe maximum water temperature limit of 85°F.

Eichna, et al (1945) carried out an extensive study to determine the upper heat limits for acclimatized working men. In applying this study to water recreations, one can assume that the information developed for a completely water saturated air environment would be applicable to a swimmer. On this basis, men are capable of prolonged hard work at temperatures between 91°F and 94°F. However, the work is inefficient, the men lose alertness and vigor and may become mild heat casualties. Moderately hard work (1200 Btu/hr) at temperatures of 94°F and above leads rapidly to total disability in most men, with excessive, and often disturbing physiologic changes. Temperatures below 91°F were considered relatively compatible environments for most men. Experience with

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military personnel exposed to warm water continuously over several hours indicates that 85°F is a safe maximum limit (NTAC, 1968).

LOW TEMPERATURE

A similar set of tolerances is observed for cold water temperatures. Here the critical problem is to maintain body temperature. In cold water, body heat is lost primarily by conduction from the inner organs through the trunk. Exposure of the limbs plays a relatively minor role in overall heat loss. A lowering of the body temperature to 74 - 77°F is considered lethal.

Keatinge (1969) carried out several extensive studies on human survival in cold water. He carried out controlled experiments and also investigated the cause of death and history of the victims (124) deaths) of the cruiseship Lakonia.

In several instances where drowning was reported as the cause of death, it was obvious that exposure to cold was more probably responsible. Keatinge reports finding victims of ship sinkings in 59°F water wearing adequate life preservers, their faces held above water, who were dead in the 1 1/2 hours it took a rescue ship to reach them. He concludes that many of the victims of ship sinkings — Titanic, Lakonia, Andrea Doria, etc — actually succumb to cold exposure.

Many factors exert significant influences on the length of time individuals can survive exposure to cold water. Among some of the most important is the amount of clothing worn, obesity of the victims, and the amount of exercise. The amount of clothing worn in the water, far from hindering survival, aids considerably in preserving body heat. An unclothed man of average build is rendered helpless after 20 - 30 minutes in water at 41°F and after 1 1/2 - 2 hours at 59°F. With thick conventional clothing the corresponding times are 40 - 60 minutes at 41°F and 4 to 5 hours at 59°F.

Shipwreck victims have been found dead after 6 hours exposure at 64°F and one hour at 32°F. An individual can survive indefinitely in water at 68°F. The amount of fat an individual has increases the tolerance to cold temperatures. There are reports of well-clothed, obese men surviving many hours at 32°F. Keatinge has developed a formula based on estimating the amounts of fat an individual has in selecting candidates to become skin divers.

A surprising finding of Keatinge's studies was that, contrary to earlier opinion, exercise in the water increases the loss of body heat and correspondingly decreases survival time. This is reflected in frequent reports of drownings of expert swimmers who tried to reach shore after a sinking, while those who remained in the water near the lost ship survived until rescued.

A careful study of reported drowning cases carried out by Press, et al (1969) seems to bear out much of the above as regards survival in cold waters. They report 299 cases out of 874 drownings, or 34% occurred in waters that were listed as very cold (assumed to be below 68°F). In addition, a much higher percent of those succumbing in cold water were considered to be good swimmers.

In summary, it may be stated that man will survive in water only over a rather limited temperature range, having very little tolerance above and below this range (Table 37). Individual differences may mitigate the effects somewhat but not to the extent that is reported for some of the other factors. It is obvious from this study that the "safe" temperature range for normal recreational activites —swimming, diving, water skiing—is between 68°F and 85°F.

PROBABILITY DISTRIBUTION FOR TEMPERATURE (R_Q VECTOR)

The temperature distribution for the Ohio river, using data obtained from the same sampling stations that acquired the pesticide data, was determined. The temperatures for the time period tested ranged from 33°F to 78°F. As expected, the low points occurred in January through March. The exponential distribution was the best fit for the available data (Table 38). This distribution is presented in Figure 27. If 68°F is selected as the safe limit for swimming, the river would be available for such use only about 30% of the time. This availability probably fits in rather well with actual practice, in that it essentially encompasses only the summer months.

TABLE 37

Limits of Temperature Tolerance for Unclothed Humans in Saturated Environments (high temperatures) or Submerged in Water (low temperatures)

Temperature			Effects
°F	°C		
95 +	35		Leads to total disability of most men after one hour of moderate effort. Excessive physiological changes.
94	34		Incapable of sustained work effort. High incidence of heat casualties.
91	33		Carry out moderate work with difficulty, inefficiently, and ineffectively.
85	30	Safe recreational range	Carry out moderate work for prolonged periods of time.
68	20		Can survive almost indefinitely in water.
64	18		Reports of death after six hours in the water
59	15		Most men rendered helpless after 1 1/2-2 hours exposure.
41	5		Helpless after 20 - 30 minutes exposure.
32	0		Death in less than one hour for clothed individuals.

TABLE 38

Temperature Distribution - Ohio River Water

Name of Distribution	"d" Statistic	Probability Data came from Cited Distribution
Normal	0.190	0.461
Lognormal	0.194	0.442
Exponential	0.185	0.498
Gamma	0.212	0.331
Weibull	0.215	0.316

Conclusion: It is not unreasonable to assume that the exponential distribution ($\theta = 6.5158 \times 10^{-2}$) best represents the universe from which the data were sampled.

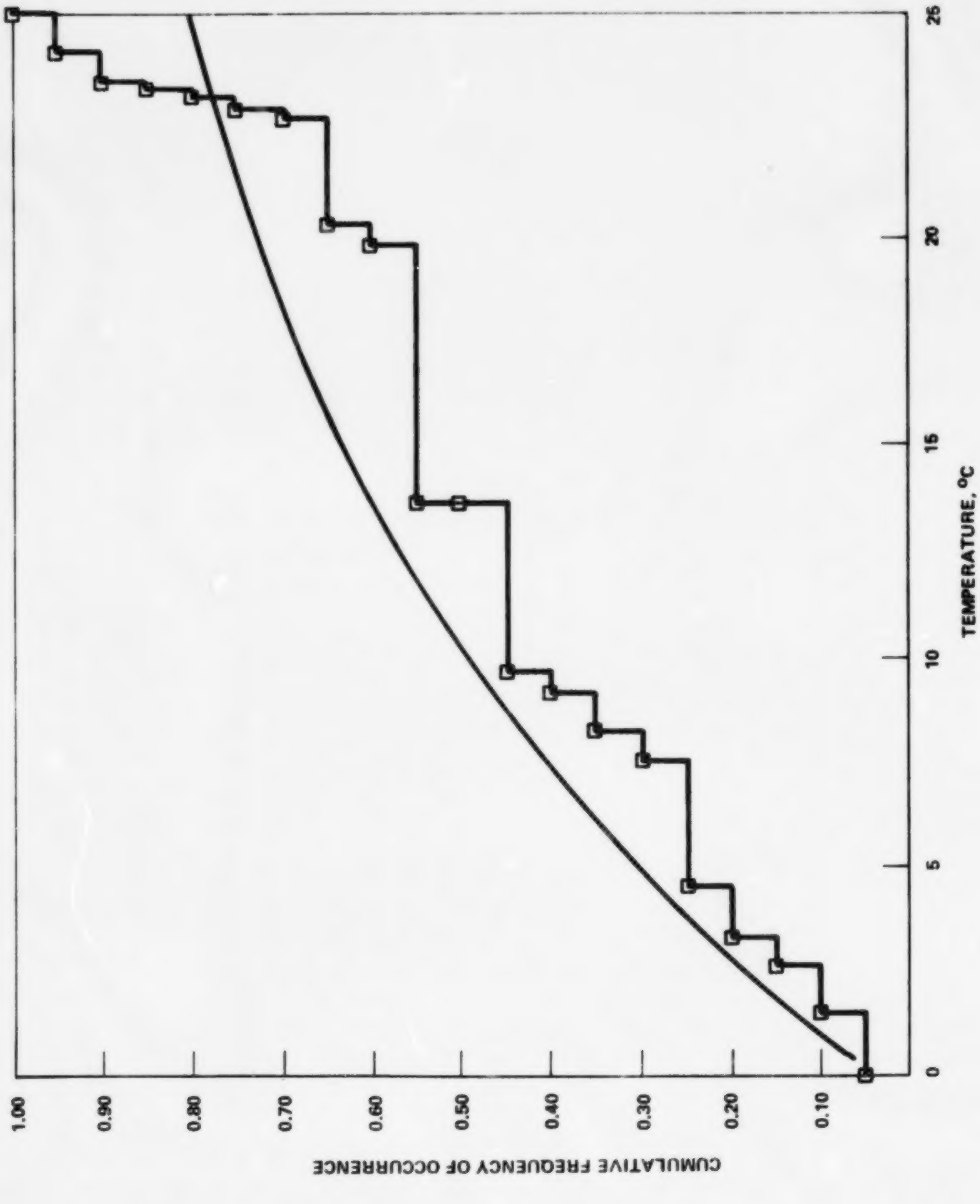

Figure 27. Temperature Distribution for the Ohio River

SECTION XI

OILS AS FACTORS OF RECREATIONAL WATER QUALITY

Oily substances that might be found in surface waters are derived from three sources: petroleum and its refined or waste products; animal and vegetable fats, usually discharged with sewage effluents; and essential oils, consisting of terpenes, aliphatic alcohols, aldehydes, ketones and lactones.

In general, there is a lack of toxicological data in the literature on the effects of ingestion or dermal sensitivity of such substances for humans exposed to them during primary contact recreation. The following summary presents the results of analogous studies, demonstrating the potential hazards that may exist, provided the required conditions of factor concentration and persistence are present.

GENERAL TOXICOLOGY

Crude petroleum is a complex mixture of saturated aliphatic and alicyclic, aromatic mono-and polycyclic, and heterocyclic compounds. The various types are classified according to the predominant component or distillation residue as paraffinic, naphthenic, aromatic, or asphaltic.

Toxicity of industrial solvents, particularly benzene, has been reviewed by Candura (1968), and of mineral oils to the skin by Lajhancova (1968). Organic solvents are capable of dissolving sebum and of penetrating the skin. Solvent dermatitis is a common industrial hazard. Solvents can enter the dermal lymphatics through fissures and lacerations and cause chemical lymphangiitis. Benzene, toluene, and xylene may cause severe toxic systemic effects when absorbed through the skin or inhaled (Fisher, 1967). Older people with dry skin are particularly prone to the effects of solvents. It is not uncommon to observe people who work with solvents to have been engaged in their particular occupation for two or more decades and then sustain cutaneous injury. The insoluble oils that are used to lubricate cutting tools and that contain petroleum oil, small amounts of vegetable oil, chlorine compounds, sulfur, and inhibitors are responsible for most cases (Pillsbury, 1957).

If ingested, gasoline may cause inebriation, vomiting, vertigo, fever, drowsiness, confusion, and cyanosis. On inhalation it can cause intense burning in the throat and lungs and possibly bronchopneumonia (Stavinoha, 1966).

The acute oral LD_{50} for rabbits fed kerosene is 28 g/kg. In humans, defatting of the skin can lead to irritation and infection. Inhalation of high concentrations of kerosene will cause headache, drowsiness, and even coma.

Mineral oil or liquid paraffin is a colorless, oily liquid that is practically tasteless and odorless even when warmed, and is insoluble in water.

Aspiration may cause lipoid pneumonia (Stavinoha, 1966).

Cyclohexane, a naphthene, is insoluble in water and has a pungent solvent odor, especially when impure. The lethal atmospheric concentration in air for mice is about 2 percent (vol. /vol.). In humans, high concentrations may act as a narcotic and skin irritant (Stavinoha, 1966).

The acute oral LD_{50} of benzene in rats is 5.7 g/kg. It has been used medicinally in treating leukemia, polycythemia vera, and malignant lymphoma. Acute toxicity symptoms in humans are irritation of mucous membranes, restlessness, convulsions, excitement, and depression. Death may follow from respiratory failure. Chronic symptoms are bone marrow depression, aplasia, and (rarely) leukemia. It may be absorbed in harmful amounts through the skin (Stavinoha, 1966). The alkylbenzenes such as toluene, xylene, and mesitylene are less toxic than benzene.

Biphenyl is used on oranges as a fungistat. The acute oral LD_{50} in rats is 2.2 g/kg. In experimental animals, CNS depression, paralysis, and convulsions have been observed on administration of biphenyl (Stavinoha, 1966).

Naphthalene occurs in the higher boiling fractions of aromatic petroleums (Dunstan, 1958). It is ordinarily obtained from coal tar, in which it is the most abundant constituent. Poisoning may occur by ingestion of large doses. Symptoms are nausea, vomiting, headache, diaphoresis, hematuria, hemolytic anemia, fever, hepatic necrosis, convulsions, and coma. No LD_{50} is given (Stavinoha, 1966) for mammals.

Anthracene and phenanthrene are 3-ring condensed aromatic hydrocarbons found in coal tar and combusion products. Phenanthrene can cause photosensitization of skin and is considered a potential carcinogen (Stavinoha, 1966). Compounds with four or more highly condensed rings such as pyrene, benzopyrene, perylene, and dibenzanthracene are carcinogenic. No data were found on the acute toxicity of these compounds.

Skin absorption rates for toluene, toluene-water mixtures (200-600 mg/l), styrene, styrene-water mixtures (66.5-269 mg/l), and xylene have been found to be, respectively, 14-23, 0.175-0.6, 9-15, 0.04-018, and 4.5-9.6 mg/cm^2 - hr (Dutkiewicz, 1968). The same author dosed rats under anesthesia orally with 0.2 ml of n-hexane or n-octane. Convulsion and death from asphyxiation occurred within a few seconds after hydrocarbon entered the lungs following volatilization in the stomach. Cardiac arrest, respiratory paralysis, and asphyxia rather than pulmonary edema or hemorrhage were found to be the causes of death. The hydrocarbons are sufficiently volatile to evaporate at body temperature and displace air in the lungs. With higher (C_{10}-C_{14}) hydrocarbons, death is due to progressive pulmonary edema and hemorrhage and occurrs after several hours. There is a sharp break in mortality between C_{14} and C_{16} compounds. The difference in response may be due to differences in the rate of spread in deeper lung structures, since the viscosities are similar and all are readily aspirated. Olefinic and acetylenic hydrocarbons give similar results.

Lengthening of the alkyl chain in alkylbenzenes decreases their toxicity to the endothelium. With petroleum distillates, mortality decreases sharply with an increase in viscosity from 39 to 59 SSU at 100°C. Hydrocarbon aerosols sprayed directly into the mouth do not present the same aspiration hazard as the liquid because the droplets do not coalesce to form a pool. It is generally held that if a child lives 24 hours after aspirating a hydrocarbon substance, he is then out of danger (Gerarde, 1963).

In South Africa, the most common cause of chemical reaction in the lungs of infants and children is paraffin, which is widely used for cooking, lighting, and heating. In a series of 61 patients ranging in age from 9 months to 19 years of age, 20 underwent X-ray examination of the chest when first seen and of these 12 showed lesions, mostly in the lower lobes. Of 10 patients admitted to the hospital, all improved rapidly and were discharged within three to seven days. There were no deaths. There was no evidence of lasting changes in the lungs in thirty of these patients a year after the paraffin ingestion occurred (Kossick, 1961).

Inhalation of air containing 50-200 mg/m^3 of xylene by mice over a period of 12 months increased erythrocytes, hemoglobin, total plasma protein, urinary excretion of 17-ketosteroids, and activity of the acetylcholine-mediating system. Decompensation occurred during the final four months. Xylene is apparently highly toxic and present permissible concentrations should be decreased (Kashin, 1968).

Median atmospheric lethal concentrations in mg/l of various hydrocarbons to mice were found to be isoprene, 157; butadiene, 270; isobutylene, 415; butane, 680; 2-methyl-1-pentene, 127; 2-methyl-2-pentene, 130; and styrene, 21.0; for rats, isoprene, 180; butadiene, 285; isobutylene, 620; butane, 658; 2-methyl-1-pentene, 115; 2-methyl-2-pentene, 114; and styrene, 118. Exposure periods were two hours for mice and four hours for rats. There was a distinct correlation between brain concentrations and toxicity for all substances studied. In cats, concentrations of hydrocarbon were highest in nervous tissue containing white matter, and in perinephric and hypodermic fat. Presence of hydrocarbon in the medulla oblongata results in respiratory arrest and death in acute intoxication (Shugaev, 1969).

CARCINOGENIC EFFECTS

Although fractions of catalytically cracked petroleum boiling above 371°C have been shown to be carcinogenic to mice by skin application, a study of paired groups of 1077 exposed and unexposed employees of three refineries showed no relation of degree of exposure to occurrence of skin tumors. Skin tumors occurred in four "potentially exposed" subjects. It is questionable whether data on skin cancer in mice can be extrapolated to humans. During the 12-year observation period, 45 neoplasms occurred in the control group and 46 in the exposed group (Wade, 1963). In a similar study of 462 asphalt workers in petroleum refineries and 379 controls, one case of lung cancer occurred in the control group and skin cancer in two workers and four controls. Carcinoma of the stomach occurred in one worker and carcinoma of the colon in a control. No cases of lung disease were classed as advanced, severe, or incapacitating. Dermatitis that occurred was localized and transient. In surveys of highway construction, roofing, trucking, and insurance industries, State Boards of Health, and Highway Commissions, only five cases of illness were attributed to asphalt contact, and none involved tumors (Baylor, 1968).

The carcinogenicity of benzo (α) pyrene and benzo (α) anthracene applied to the skin of mice was enhanced by a factor of 1000 when n-dodecane was used as solvent in comparison to carcinogenicity of the same substances in decalin. The carcinogenicity was also increased when n-dodecanol or 1-phenyldodecane was used as solvent. These results indicate that certain long-chain hydrocarbons may play the decisive role in determinging the carcinogenic potency of a mixture. The importance of the concentration of the initiator may be relatively minor although its presence in minute amounts is apparently necessary (Bingham, 1969).

SUMMARY

Contamination of recreational waters with oily substances may occur as a result of oil drilling operations in coastal waters, seepage from natural underwater oil deposits, discharge of fuel tank contents of ships, either accidentally or deliberately, and discharge of industrial wastes. Although the presence of oily substances would make the water esthetically unattractive because of odor and fouling of equipment and bodies of bathers, and the possibility exists that recreationists might still use the water. Toxicity of oily substances by ingestion or skin absorption or by inhalation of vapors is relatively low except in the case of aromatics. The possibility of lung injury following ingestion of a gross amount of floating crude petroleum followed by vomiting and aspiration into the lungs must be considered. Skin irritation, either caused directly by contact with the oil or by the use of petroleum solvents to remove it, is also a factor. Skin irritation by oil is increased in mixtures with brine (Gage, 1963). Light monolayer films which may not be visually detectable could coat the entire body of a swimmer entering the water. These could magnify toxicity effects.

SECTION XII

CHEMICAL AND PHYSICAL FACTORS OF RECREATIONAL WATER QUALITY

The amount of information available relating the factors of pH, temperature, and clarity to ill-effect in recreation water was found to be more limited than for the other factors of interest. Consequently, these items have been combined in a single section. In general, it may be stated that the criteria recommended by the National Technical Advisory Committee (NTAC) on Water Quality Criteria represent the most likely conclusions to be drawn from available data, and the views represented by the group have been largely used as the basis for this section.

E. W. Mood (NTAC, 1968) has reviewed the literature on the relation between pH and aquatic activity, and explored the bases for the establishment of criteria for those properties of water that may cause eye irritation to bathers and swimmers. Since his review contains a concise and useful account of the relation of pH to recreation water, and little supplemental information was encountered in our survey, its major points are brought out below.

Knowledge about the characteristics of water that may cause irritation to the eyes of swimmers has been developed through research efforts of ophthalmologists and others in connection with investigations on the preparation of ophthalmic solutions. Since the ideal non-irritating solution should have physico-chemical properties similar to tears, studies were undertaken initially to determine the chemical composition of lacrimal fluid, particularly of its hydrogen-ion concentration, or pH, its buffer capacity, and its toxicity. Although early studies of the hydrogen-ion concentration of tears revealed values ranging from 6. 3 to 8. 6, Hind and Goyan (1947; 1949) found that lacrimal fluid has a pH of approximately 7. 4.

Lacrimal fluid is only weakly buffered and has the capacity to bring the pH of an unbuffered solution from as low as 3. 5 or as high as 10. 5 to within tolerable limits in a very short time. However, if the pH of a solution in direct contact with the eyes is lower than 7. 3 or higher than 7. 5, pain may be elicited.

Since the most sensitive part of the body that may come in contact with a given water is the eye, the toxicity or salt concentration is another important aspect. Early studies by Hind and Goyan (1947) showed that a sodium chloride equivalent range of 0. 5 to 2. 0 percent concentration was well tolerted. Later, Riegelmann, et al (1955) and Riegelmann and Vaughan (1958) suggested that the range be narrowed to the equivalence of between 0. 7 to 1. 5 percent sodium chloride.

Seawater has a sodium chloride equivalent tonicity of 3. 5 percent. It is mildly irritating to most swimmers but in normal recreational activity, exposure is limited. Concentrations higher than 3.5 percent NaCl are rarely encountered. Thus, Mood (1968) concludes that tonicity of recreation waters is of much less importance than the hydrogen-ion concentration and the buffer capacity in preventing or reducing eye irritation to bathers and swimmers.

"In summary, when water quality standards are proposed for swimming, bathing, and other similar uses, consideration should be given to those physico-chemical properties that may cause or contribute to eye irritation, if principal importance is the hydrogen-ion concentration with codependence upon the buffer capacity of the water. Ideally, the pH of the water should be approximately the same as lacrimal fluid, which is about 7.4 for most people; a range of pH values from 6.5 to 8.3 can be tolerated under average conditions. If the recreation water is relatively free of dissolved solids and has a very low buffer capacity, pH values from 5.0 to 9.0 should be acceptable. However, for recreation water having pH less than 6.5 or greater than 8.3, waste discharge standards should include prohibition against releases that will increase the buffer capacity of the receiving waters and yet maintain the pH below 6.5 or greater than 8.3. Tonicity standards do not seem to have any practical value " (NTAC, 1968).

The distribution of pH values for the Ohio River, using data from the same sampling stations and over the same time periods as for the pesticide and temperature studies, was analyzed. The Weibull distribution most closely approximated the sampled universe (Table 39); this is represented in Figure 28. The pH values ranged from 6.8 to 7.7. These are well within the values that can be safely tolerated by recreationists.

CLARITY (TURBIDITY)

Clarity in recreational waters is highly desirable from the standpoint of visual appeal, recreational enjoyment, and safety. Variation in natural conditions make it difficult to set absolute criteria for this factor. However, natural conditions taken into account, turbidity attributable to human activity should nonetheless be controlled in recreation waters where feasible.

For primary contact recreation waters, clarity has been recommended to be such that a Secchi disc is visible at a minimum depth of four feet. In "learn to swim" areas, the clarity should be such that a Secchi disc on the bottom is visible. In diving areas, the clarity shall equal the minimum required by safety standards, depending on the height of the diving platform or board (NTAC, 1968).

The Secchi disc is a device used to measure visibility depths in water. The upper surface of a circular metal plate, 20 centimeters in diameter, is divided into four quadrants and so painted that two quadrants directly opposite each other are black and the intervening ones white. When suspended to various depths of water by means of a graduated line,

TABLE 39

pH Concentration Distribution - Ohio River Water

Name of Distribution	"d" Statistic	Probability Data came from Cited Distribution
Normal	0.1256	0.910
Lognormal	0.118	0.942
Exponential	0.595	0.000
Gamma	0.164	0.656
Weibull	0.107	0.976

Conclusion: It is not unreasonable to assume that the Weibull distribution ($\hat{c} = 6.7135$, $\hat{\theta} = 4.9604 \times 10^{-1}$, $\hat{k} = 1.7902$) best represents the universe from which the data were sampled.

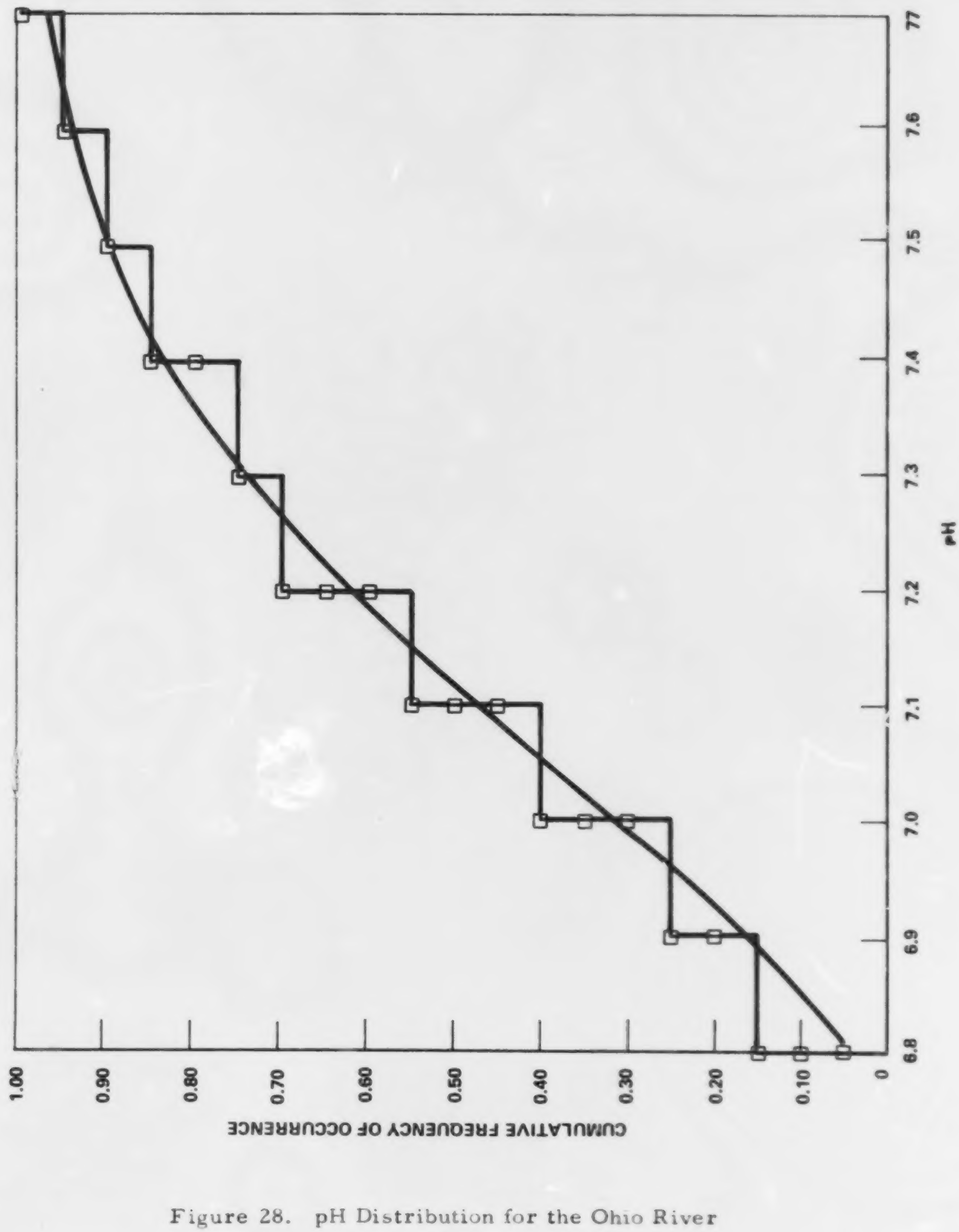

Figure 28. pH Distribution for the Ohio River

its point of disappearance indicates the limit of visibility. If it is then raised until it reappears and the average of the two depths is taken as the Secchi disc transparency.

Light penetration into waters is extremely variable in different water bodies. Clarke (1939) pointed out that the diminution of the intensity of light in its passage through water follows a definite mathematical formula. The relationship between the depth of water and the amount of light penetrating to that depth is semilogarithmic. Even the clearest waters impede the passage of light to some extent; light passed through 100 meters of distilled water is reduced in intensity to one to two percent of its incident value.

The principal factors affecting the depth of light penetration in natural waters include suspended microscopic plants and animals, suspended mineral particles, stains that impart a color, detergent foams, dense mats of floating and suspended debris, or a combination of these factors.

Beeton (1958) made 57 paired photometer and Secchi disc measurements at 18 stations in Saginaw Bay in Lake Huron. He found that the average percentage transmission of surface light introduced, at the Secchi disc depth, was 14.7 percent. Verduin (1956) made simultaneous determinations with the Secchi disc and submarine photometer during August 1955, on Lake Erie. The Secchi disc readings in meters were plotted against the depth associated with one percent of the surface light. A line drawn by inspection through the scatter diagram suggests that an approximate estimation of the euphotic zone can be obtained by multiplying the Secchi disc readings by five. Riley (1941) used a factor of three. Verduin (1956) computed a factor of 2.5 using the data of Bursch (1955). Rawson (1950) lists a factor of 4.3 when the Secchi disc reading is about one meter.

The maximum Secchi disc reading reported for Lake Tahoe, California-Nevada, was 136 feet at one station on April 4, 1962 (McGauhey, et al 1963). A minimum Secchi disc reading of 49 feet was recorded in Emerald Bay of Lake Tahoe on May 21, 1962. In contrast, the Secchi disc disappeared in three feet in Lake Sebasticooke, Maine, during a July 1965 study. In areas with less dense algal growths, the readings were increased to eight feet. Beetan (1965) recorded the average Secchi disc depth for Lake Superior as 32.5 feet; Lake Michigan, 19.6 feet; and Lake Erie, 14.6 feet (Mackenthun and Ingram, 1967).

The Jackson candle turbidimeter (Standard Methods for the Examination of Water and Wastewater, 12th Edition, 1965) is the standard instrument for making measurements of turbidity. Field determinations are made with direct reading colorimeters calibrated for this test and results are expressed as Jackson Turbidity Units (JTU).

Buck (1955), investigating various fishery waters, observed that maximum production occurred in farm ponds where the average turbidity was less than 25 JTU. Between 25 and 100 JTU, fish yield dropped and in muddy ponds of over 100 JTU, the yield was only 18 percent of clear ponds.

Walton (1961) analyzed data from three waterworks treating water by chlorination only. Coliform organisms were detected in one water that averaged 10 JTU with an occasional reading of 100 JTU. No coliforms were detected in the chlorinated waters with 0 to 5 JTU.

Wang and Brabec (1969) reported on the "Nature of Turbidity in the Illinois River". They presented a graph tht related Secchi disc visibility to water turbidity in JTU. This is presented in Figure 29.

An analysis of the turbidity readings along the sampling points of the Ohio River, as previously discussed, is presented in Table 40. The lognormal distribution presented in Figure 30 shows the very wide range of turbidities (15 to 750 scale units) within these data.

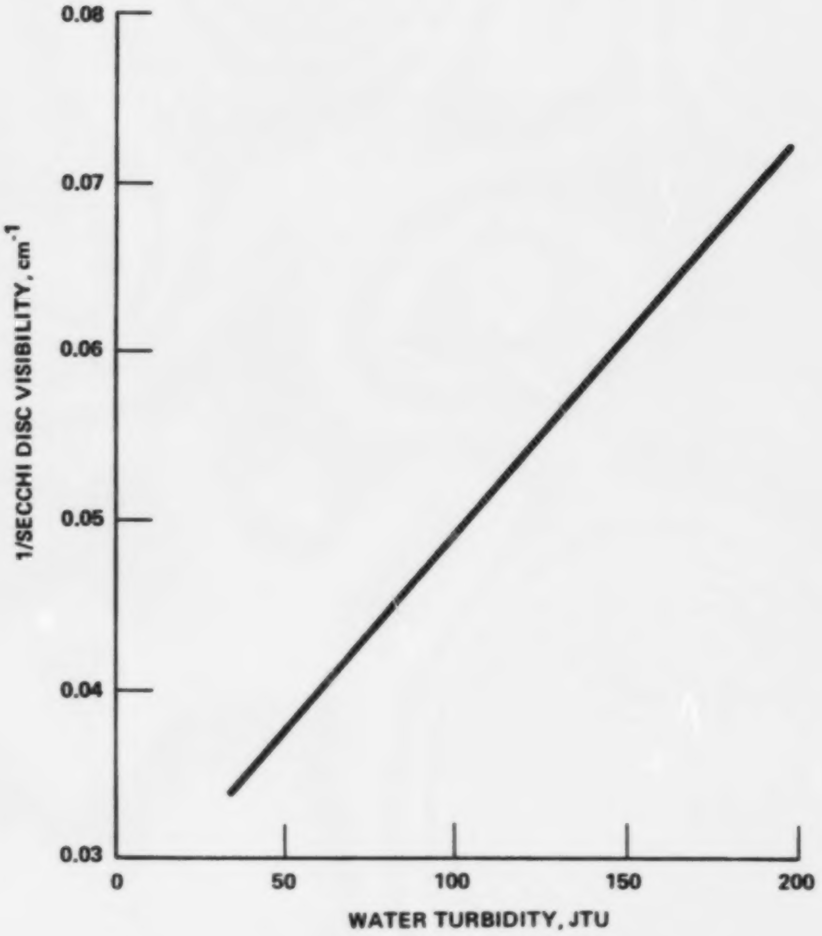

Figure 29. Relationship Between Secchi Disc Visibility and Water Turbidity in the Illinois River

TABLE 40

Turbidity Distribution - Ohio River Water

Name of Distribution	"d" Statistic	Probability Date came from Cited Distribution
Normal	0.260	0.134
Lognormal	0.104	0.981
Exponential	0.178	0.548
Gamma	0.125	0.913
Weibull	0.223	0.271

Conclusion: It is not unreasonable to assume that the Lognormal distribution ($\hat{y}$ = 4.4566, $\hat{\sigma}y$ - 1.0928) best represents the universe from which the data were sampled.

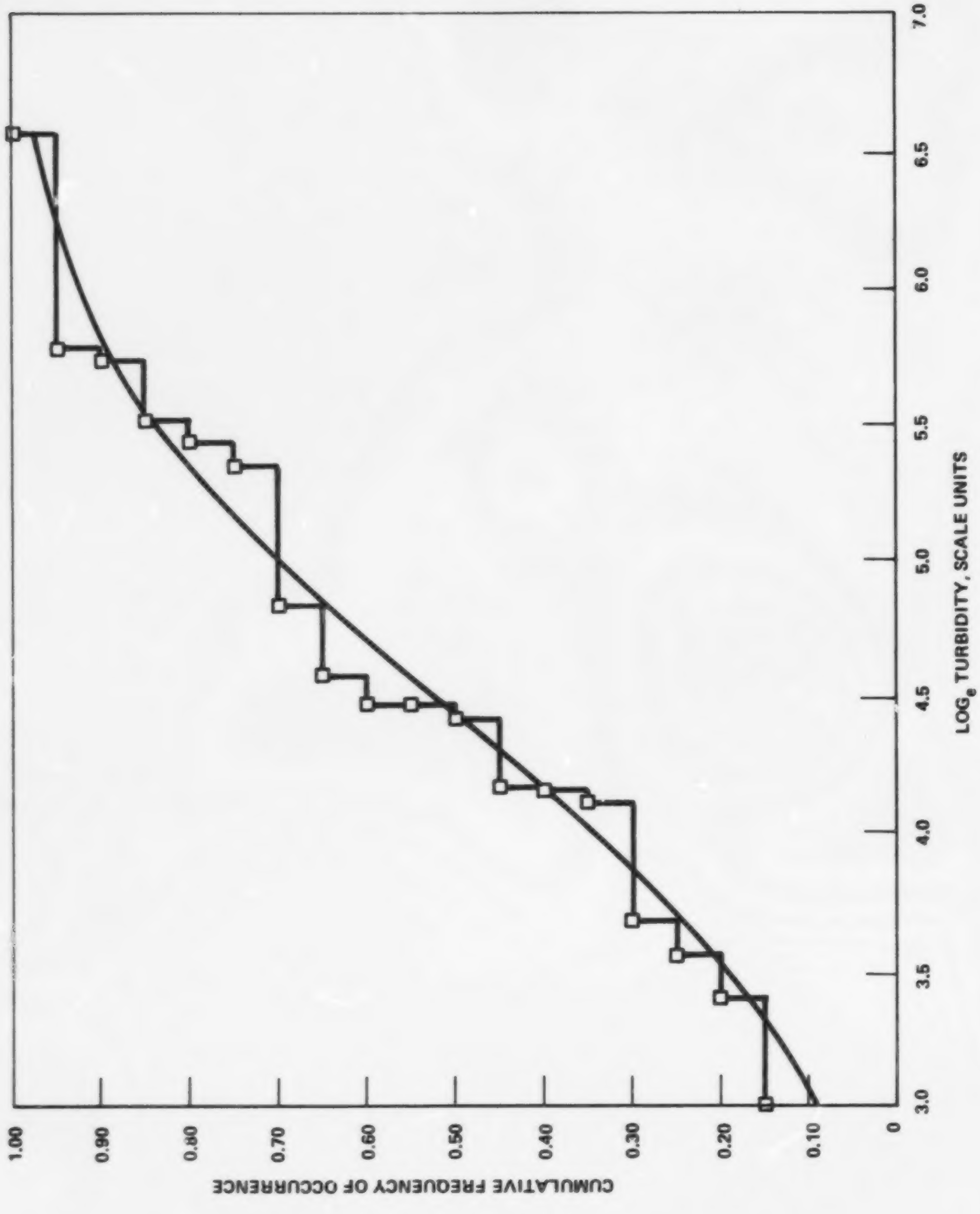

Figure 30. Turbidity Distribution for the Ohio River

SECTION XIII

ACKNOWLEDGMENTS

This program was carried out under the direction of Dr. Byron J. Mechalas as program manager. Dr. K. K. Hekimian was responsible for the development of the conversion methodology and was ably assisted by Envirogenics Consultant Dr. R. H. Dudley, who developed the mathematical model and the computor techniques used in the program.

Dr. Lewis A. Schinazi carried out the search of the literature, and preparation of abstracts for documenting the data base. Dr. Carl Rambow (now deceased) of Montgomery Research was a subcontractor for the survey of pesticides and state standards.

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SECTION XIV

APPENDIX A

INTRODUCTION

To investigate current criteria and promulgated standards for recreational waters, a letter of inquiry was sent to fifty states and eight territories and districts. This survey was conducted by Montgomery Research, Inc., Pasadena, California, subcontractor to The Envirogenics Co. on this program. The text of the letter used is as follows:

> "Our firm, together with Aerojet-General Corporation of El Monte, has contracted with the Federal Water Pollution Control Administration to evaluate the relationships between (a) certain water quality parameters, and (b) recreational uses of water. Part of this study involves review of state standards for recreational water quality for those parameters listed below:
>
> Total coliforms
> Fecal coliforms
> Viruses
> Salmonella
> Oils
> pH
> Temperature
> Clarity
> Pesticides
>
> I would appreciate receiving copies of any documents giving limiting values of these parameters as adopted by your state, the date of adoption, and any pertinent remarks you may wish to make. Particularly desired are the bases on which the limitations were established, or their sources.
>
> I realize that some of this information is contained in the water quality standards submitted to the United States Government in compliance with the Clean Water Act. However, in most cases it is impossible to derive the needed information from these Standards because they apply to many different bodies of water of which few, if any, are used for recreation and no other purpose. It, therefore, becomes necessary to request this information directly.
>
> Your cooperation in providing this information will be greatly appreciated. It will enable us to complete our study and derive conclusions that will be meaningful and useful in the future in establishing or modifying criteria."

Replies were received from the fifty states and six of the districts in the form of published water quality documents. After a thorough review of the documents, the data relevant to the factors of interest were tabulated and are compared in Table 41. Summaries of data on the various factors have been presented as Tables 1-8 in the main body of the text.

The various documents from which the data were derived have been listed in Bibliography C of Section XV.

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TABLE 41

Summary State Recreational Water Quality Criteria

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Alabama	Water Improvement Commission	May 5, 1967	No standard set	1000/100 ml as a monthly average during May through September, nor exceed this value in any two consecutive samples collected during these months.	6.0-8.5	More than 10% by addition of wastes nor shall these wastes cause the temperature to exceed 90°F.	Non-quantitative[a]	Non-quantitative	Criteria based on present scientific knowledge, experience and judgement
Alaska	Department of Health and Welfare	June 20, 1967 Revised November 10, 1967	1000/100 ml average, with 20% of samples not to exceed this number. No sample shall exceed 2400/100 ml.	No standard set	6.5-8.5	Numerical standard not set.	Non-quantitative[a]	Non-quantitative	Public hearings were held and all reports, written and verbal were reviewed before standards adopted.
Arizona	State Department of Health, Division of Water Pollution Control	July, 1968	No standard set	A geometric mean of 200/100 ml nor shall more than 10% of the total samples during any 30-day period exceed 400/100 ml. For waters other than primary contact value shall not exceed a geometric mean of 1000/100 ml nor shall more than 10% of the samples during any 30-day period exceed 2000/100 ml.	6.5-8.6	Maximum 93°F	Non-quantitative[a]	50 JU warm water streams; 10 JU cold water streams; 25 JU warm water lakes; 10 JU cold water lakes.	Basis for present criteria is considerations of present conditions, contributing factors and quality requirements for specified uses.

[a] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Arkansas	Pollution Control Commission	July 23, 1964	200/100 ml in more than 20% of the samples tested, nor shall any of the samples contain more than 500/100 ml.	No standard set	6.0-9.0	No standard set	The level of toxic substances shall not exceed 0.1 of 48-hr median tolerance level.	Non-quantitative[1]	Waters covered by criteria are Interstate streams any bathing place at a lake, pond or stream.
California	Department of Public Health, Bureau of Sanitary Engineering	1958	1000/100 ml, provided that not more than 20% of the samples at any sampling station in any 30-day period, may exceed 1000/100 ml, and provided further that no single sample when verified by a repeat sample taken within 48 hrs shall exceed 10,000/100 ml.	No standard set	No standard set	No standard set	Non-quantitative[1]	Non-quantitative[1]	Waters covered by criteria are public beaches and public water contact sports areas of the ocean waters and bays.
Colorado	Department of Public Health, Water Pollution Control Division	March 1, 1967	1000/100 ml as a monthly average nor exceed this number in more than 20% of the samples examined during any month nor exceed 2400/100 ml in a single sample.	100/100 ml and fecal streptococcus count shall not exceed 20/100 ml. Both of these limits to be an average of 5 consecutive samples within a month.	6.5-8.5	No standard set	Non-quantitative[1]	Non-quantitative[1]	Basic standards were prepared after a number of conferences with the 7 States located within the Colorado River Basin. Standards for body contact sports based on recommendations of Federal Authorities. All Colorado surface waters are classified for body contact sports.

[1] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Connecticut	State Department of Health, Environmental Health Services Division	Not given	A median of 1000/100 ml nor more than 2400 in more than 20% of the samples collected.	No standard set	6.5-8.0	Not to exceed 85°F or raise the normal temperature of the receiving water more than 4°F.	Non-quantitative[a]	Non-quantitative[a]	Standards were guided by recommendations which appear in "Report of the Committee on Water Quality Criteria".
Delaware	State Board of Health, Bureau of Environmental Health	March, 1968	1000/100 ml during any month of the recreation season; nor exceed this number in more than 20% of the samples examined during any such month; nor exceed 2400/100 ml on any day in areas designated by the Commission for water contact recreation.	No standard set	6.5-8.5	Not to exceed 5°F above normal for the locality, with maximums specified for given waters.	Non-quantitative[a]	Non-quantitative[a]	Standards recommended by the FWPCA for recreational waters were adopted. All waters of the state covered by criteria
Delaware River Basin	Delaware River Basin Commission, Water Quality Branch	March, 1968	No standard set	Effluent standard effective disinfection means the treatment of wastes such that the number of organisms remaining after treatment does not exceed 200/100 ml as a geometric average, nor 1000/100 ml in more than 10% of the samples taken over a period of 30 consecutive days.	6.0-8.5	Shall not exceed 5°F above the average daily temperature gradient displayed during 1961-66 period, or a maximum of 86°F.	Non-quantitative[a]	Maximum monthly mean 40 units, maximum 150 units	Not given.
District of Columbia	Department of Public Health, Water Quality Control Division	January 17, 1969	No standard set	240/100 ml in 90% of the samples collected each month. (not applicable during or immediately following periods of rainfall)	6.0-8.5	Not to exceed 90°F. No increase in natural water temperature caused by artificial heat inputs shall exceed 5°F after reasonable mixing.	Non-quantitative[a]	Non-quantitative[a]	Numerical values of the criteria were principally derived from the report of the National Technical Advisory Committee on Water Quality Criteria.

[a] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Florida	Department of Health and Rehabilitative Services	None given	1000/100 ml Survey of natural bathing areas shall consist of a minimum of three bacteriological samples collected from the proposed bathing area daily for the first three days of each week for three consecutive weeks.	No standard set	No standard set	No standard set	No standard set	No standard set	Not given
Georgia	State Water Quality Control Board	Currently under review	No standard set	Not to exceed a mean of 1,000 per 100 ml based on at least four samples taken over a 30-day period and not to exceed 4,000 per 100 ml in more than 5 percent of the samples taken in any 90-day period.	6.0-8.5 (swamp waters may have pH of 4.5)	Presently being studied. Current standard is a max. of 93.2°F with a max. of 10°F rise above natural but below 93.2°F allowable.	Non-quantitative*	Non-quantitative*	The State of Georgia has complated a study with the FWPCA on fecal coliform standards. This study will probably result in the following fecal coliform standards being adopted: Marine waters: 100/100 ml MPN Reservoirs: 300/100 ml Streams: 500/100 ml
Guam	Water Pollution Control Commission	April, 1968	No standard set	Shall not exceed an arithmetic mean of 200/100 ml nor exceed 400/100 ml in more than 10% of samples during any 30-day period.	7.0-8.3	Shall not exceed 85°F due to influence of other than natural conditions.	Non-quantitative*	Visibility shall not be reduced by more than 10% of normal values as measured by secchi disc	Not given

*The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Hawaii	Department of Health	January 26, 1968	Median not to exceed 1000/100 ml nor shall more than 10% of the samples exceed 2400/100 ml.	Arithmetic average of 200/100 ml during any 30-day period nor shall more than 10% of the samples exceed 400/100 ml in the same time period	Should not vary more than 1/2 unit from natural conditions but not lower than 7.0 nor higher than 8.5 from other than natural causes.	Temperature of receiving waters shall not change more than 1.5°F from natural conditions	Non-quantitative	Secchi disc or secchi disc equivalent as "extinction coefficient" determinators shall not be altered from natural conditions more than 10%.	Criteria based upon the best currently available data.
Idaho	State Board of Health	September, 1968	240/100 ml with 20% of the samples not to exceed 1000/100 ml.	50/100 ml with 20% of the samples not to exceed 200/100 ml	6.5-9.0 Induced variation not to be more than 0.5 unit.	No measurable increase when stream temperatures are 66°F or above or more than 2°F when stream temperatures are 64°F or less.	Non-quantitative	Not to exceed 5 JTU	Public hearings were held with testimony received on the proposed water quality standards. Testimony given at these hearings was considered and utilized where possible in the development of the final standards.

The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Illinois	Sanitary Water Board	March 7, 1967	5000/100 ml as a monthly average, nor exceed this number in more than 20% of the samples examined during any month, nor exceed 20,000/100 ml in more than 5% of such samples	For primary contact recreation – shall not exceed a geometric mean of 200/100 ml, nor shall more than 100% of the total samples during any 30-day period exceed 400/100 ml. For secondary contact recreation – shall not exceed a geometric mean of 1000/100 ml nor shall they equal or exceed 2000/100 ml in more than 10% of the samples.	Not specifically given for recreational waters.	Not specifically given for recreational waters.	Non-quantitative[*]	Non-quantitative[*]	Not given.
Indiana	Stream Pollution Control Board	June 11, 1967	1000/100 ml as a monthly average during any month of the recreational season, nor exceed this number in more than 20% of the samples examined during any month of the recreational season, nor exceed 2400/100 ml on any day.	No standard set	No standard set	No standard set	Non-quantitative[*]	Non-quantitative[*]	All reservoirs and lakes shall be maintained for whole body contact recreation. Streams to be protected are the Ohio River, Wabash River where it forms a boundary with Illinois, the St. Joseph River in Elkart & St. Joseph Counties & St. Joseph River in Allen County.

[*] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not be Increased	Pesticides and Oils	Toxics	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Iowa	Water Pollution Control Commission State Dept. of Health	1968	1,000/100 ml as a monthly average nor exceed this value in more than 20% of the samples in any one month nor exceed 2,400/100 ml in any one sample. These figures are used as guides until suitable indices are developed.	No standard set	6.8-9.0	Not to exceed 93°F during May through Nov., and not to exceed 73°F during Dec. through April.	Non-quantitative	Non-quantitative	Information received by other state agencies presentations at public hearings was used by the Commission for establishing recreational standards. Coliform standards are used as guides only, as studies have shown high bacterial concentrations associated with land runoff, and public health studies to date have shown little direct correlation between coliform concentrations and water-borne diseases.

The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Kansas	Department of Health, Environmental Health Services	January 1, 1968	1000/100 ml - Survey work and professional evaluation are used to the greatest extent possible.	No standard set	6.5-8.5	No standard set	Non-quantitative[a]	Non-quantitative[a]	Not given.
Kentucky	Water Pollution Control Commission	June 9, 1969	1000/100 ml as a monthly arithmetical average nor exceed this number in more than 20% of the samples examined during any month, nor exceed 2400/100 ml on any day.	No standard set	No values below 5.0 nor above 9.0 and preferably between 6.5 and 8.5 (excepted from Dept. of Interior approval to be revised).	Not to exceed 93°F at any time during the months of May through November & not to exceed 73°F at any time December through April (to be revised)	Non-quantitative[a]	Non-quantitative[a]	When these standards were initiated the Kentucky Water Pollution Control Commission recognized the ORSANCO criteria with slight modifications as the basis of their proposed criteria
Louisiana	Department of Health, Bureau of Environmental Health	1968	There is a varying range for recreational waters Range is from 70/100 ml for some waters, 230/100 ml for some waters, 542/100 ml for some waters, and 1600/100 ml for majority of waters.	No standard set	Values vary for different waters range-6.5-9.5	Not to be raised more than 3°C above normal ambient water temperature, nor to exceed 36°C.	Non-quantitative[a]	Non-quantitative[a]	Criteria based on present and potential uses of Louisiana waters and the existing water quality indicated in data accumulated through monitoring programs of various agencies.

[a] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Maine	Water and Air Environmental Improvement Commission	October 7, 1967	No single standard sample collected from a public bathing beach shall show a maximum of over 10 confirmed B. Coli per ml. When a single sample shows over 10, but less than 30 per ml, a repeat sample shall be immediately taken. If any single sample shows 30 or more B. Coli per ml, the beach shall be closed until water quality is shown to be satisfactory.	No standard set	No standard set	No standard set	Non-quantitative[*]	Non-quantitative[*]	Not given
Maryland	Department of Health, Division of Water and Sewerage	April, 1969	No standard set	240/100 ml. When the fecal coliform organism density exceeds this value the bacterial quality shall be considered acceptable only if a second detailed sanitary survey and evaluation discloses no significant public health risk.	6.5-8.5	Maximums vary for different waters. No specific standard for recreational use.	Non-quantitative[*]	Non-quantitative[*]	Not given

[*] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Massachusetts	Water Resources Commission	February 17, 1967	1000/100 ml during any monthly sampling period, nor 2400/100 ml in more than 20% of samples examined during such period.	No standard set	6.5-8.0	No increase except where the increase will not exceed the recommended limit on the most sensitive receiving water use. In no case raise the normal water temperature more than 4°F.	Non-quantitative[a]	Non-quantitative[a]	Limits are based on New England Interstate Water Pollution Control Commission standards
Michigan	Department of Natural Resources	Interstate standards adopted June 28, 1967 Intrastate standards adopted January 4, 1968	Total body contact- 1000/100 ml nor shall 20% of the samples examined exceed 5000. Partial body contact- 5000/100 ml nor shall 20% of the samples examined exceed 10,000.	Total body contact- geometric average for the same ten consecutive samples exceed 100/100 ml. Partial body contact- geometric average for the same ten consecutive samples shall not exceed 1000/100 ml.	Maintained within a range of 6.5-8.5 with maximum induced variation of 0.5 unit within this range.	90°F maximum	Non-quantitative[a]	Non-quantitative[a]	Standards based on information gained from review of current literature, existing standards and present water quality data, and testimony given at public hearings.
Minnesota	Minnesota Pollution Control Agency	August 3, 1967	1000/100 ml	No standard set	6.5-8.0	No standard set	Non quantitative[a]	10	Not given.

[a] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Mississippi	Air and Water Pollution Control Commission	March 1968	No standard set	1000/100 ml as a monthly average value, nor exceed this number in more than 20% of the samples examined during any month nor exceed 2400/100 ml on any day.	6.0-8.5	Shall not be increased more than 10°F above the natural prevailing background temperature, nor exceed a maximum of 93°F after reasonable mixing.	Non-quantitative*	Non-quantitative*	Standards based on good sanitary engineering practice and consultation with the affected parties.
Missouri	Water Pollution Board	June, 1968	No standard set	A geometric mean of 200/100 ml nor shall more than 10% of total samples during any 30 day period exceed 400/100 ml.	6.5-8.5	Effluents shall not elevate or depress average temperature more than 5°F. Maximum temperature due to effluent 90°F.	Non-quantitative*	Shall not exceed 20 turbidity units due to effluents.	Public hearings were held to establish the uses of the waters of the state and to gather information to be used in the development of water quality standards to protect these uses.
Montana	Department of Health, Division of Environmental Sanitation	October 5, 1967	1000/100 ml with not more than 20% of the samples exceeding this value.	No standard set	6.5-9.5	No standard set	Non-quantitative*	Increase in naturally occuring turbidity shall not exceed 10 JCU.	Not given.
Nebraska	Department of Health, Water Pollution Control Agency	January, 1969	No standard set	A geometric mean of 200/100 ml based on at least five samples per 30 day period and shall not exceed 400/100 ml in more than 10% of the samples.	6.5-9.0	Shall not exceed 5°F over normal temperature of water from May to October and not more than 10°F from November to April. Maximum rate of change limited to 2°F per hour.	Non-quantitative*	In no case shall turbidity caused by waste water impart more than a 10% increase in turbidity to the receiving water.	Standards based on testimony received at public hearings and on reflection of past experience.

*The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Nevada	Division of Health, Bureau of Environmental Health	July, 1967	1000/100 ml if Fecal Streptococci are less than 100 or 5000/100 ml if Fecal Streptococci are less than 20. (Criteria for Colorado River only, other standards set for Lake Tahoe.)	No standard set	7.0-8.5	Not more than 18°C summer or 14°C in winter, 20°C below Davis Dam.	Non-quantitative[a]	Non-quantitative[a]	"Guidelines for formulating water quality standards for the waters of the Colorado River System" are incorporated as a supplement to the standards.
New Hampshire	Water Supply and Pollution Control Commission	January, 1969	240/100 ml	No standard set	6.5-8.0	Any stream temperature increase associated with the discharge of treated sewage, waste, or cooling water shall not be such as to appreciably interfere with the uses assigned to this class.	Non-quantitative[a]	Non-quantitative[a]	Not given
New Jersey	Department of Health, Water Pollution Control	May 1, 1967	No standard set	No standard set	6.5-8.5	Non-quantitative[a]	Non-quantitative[a]	Non-quantitative[a]	It is anticipated that the water quality criteria may be amended the end of 1969.
New Mexico	Health and Social Services Department, Environmental Services Division	March, 1968	No standard set	The geometric average of five consecutive daily samples collected under similar conditions should not exceed 200/100 ml.	Not specific for waters used for body contact sports.	Not specific for recreational use of waters.	Non-quantitative[a]	Non-quantitative[a]	Not given

[a] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale an Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
New York	Department of Health	November, 1968	For Boundary waters - 2400/100 ml MPN median value.	No standard set	6.5-8.5	Streams-Non-Trout Waters Maximum 90°F with temperature change not to exceed 5°F above natural in 50% of cross-sectional area to a maximum of 85°F. Trout Waters No discharge at a temperature over 70°F. Maximum increase June through Sept. 2°F, Oct. through May 5°F over normal.	Non-quantitative*	Non-quantitative*	Not given
North Carolina	Department of Water and Air Resources	January 30, 1968	No standard set	An average of 200/100 ml on at least 5 consecutive samples examined during any 30 day period and not to exceed 400/100 ml in more than 20% of the samples examined during any 30 day period. (Applicable only May - September.)	Shall be normal for the waters of the area, which range between 6.0-8.5, except swamp waters may have a low of 4.3.	Not to exceed 7°F above the ambient stream or water temperature, and in no case exceed 95°F.	Non-quantitative*	Non-quantitative*	Standards established following public hearings.
North Dakota	Department of Health, Division of Water Supply and Pollution Control	May, 1967	1000/100 ml	No standard set	6.5-9.0	Not to exceed 90°F.	Non-quantitative*	Non-quantitative*	Adopted standards are based on the results of public hearings and following "Guidelines for Establishing Water Quality Standards for Interstate Waters."

*The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Ohio River Valley	Ohio River Valley Water Sanitation Commission	1967	No standard set	200/100 ml as a monthly geometric mean based on not less than five samples per month, nor exceed 400/100 ml in more than 10% of all samples taken during a month.	No standard set.	No standard set	Non-quantitative[*]	Non-quantitative[*]	Member states are: Illinois, Indiana, Kentucky, New York, Pennsylvania, Virginia, West Virginia and Ohio. The signatory States conducted public hearings and received testimony. Committees were established and recommendations for standards made. All member states submitted quality standards and plans for their implementation.
Ohio	- - -	1967	1000/100 ml as a monthly average nor exceed this in more than 20% of the samples examined during any month nor exceed 2400 per 100 ml on any day.	No standard set.	No standard set.	No standard set	Non quantitative[*]	Non-quantitative[*]	Not given.
Oklahoma	Department of Health, Environmental Health Services	1968	1000/100 ml as a monthly average during the recreational season, nor exceed this number in more than 20% of the samples examined during any one month, nor exceed 2400/100 ml on any day except during periods of storm run off.	A geometric mean of 200/100 ml nor shall more than 10% of the total samples during any 30 day period exceed 400/100 ml.	6.5-8.5	Changes in temperature from other than natural sources shall be limited to a maximum of 5°F with specific maximums set for various fish propagation areas.	Non-quantitative[*]	Non-quantitative[*]	Public hearings held in accordance with State statutes. Current water quality and history of State waters reviewed before standards adopted

[*] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Oregon	Oregon State Sanitary Authority	June 1, 1967	Average concentration 1000/100 ml with 20% of the samples not to exceed 2400/100 ml.	No standard set	6.5-8.5	Maximum increase permitted 2°F with specific temperature maximums stated for individual waters.	Non-quantitative*	Not to exceed 5 JTU above natural background values.	Waters covered by criteria are Goose Lake, Grande Ronde River, Walla Walla River, Columbia River, Snake River, Klamath River, and Willamette River.
Pennsylvania	Department of Health, Division of Water Quality	Not given	1000/100 ml as an arithmetic value, not to exceed 1000/100 ml in more than two consecutive samples nor exceed 2400/100 ml in more than one sample. (For the period 9/16 to 5/14 of any year not to exceed 5000/100 ml as a monthly average value, nor exceed this number in more than 20% of the samples collected during any month, nor exceed 20,000/100 ml in more than 5% of the samples.)	A log mean of 200/100 ml in any five or more samples for a 30 day period, nor shall more than 10% of the total samples during any 30 day period show a density of more than 400/100 ml.	No standard set solely for recreational water protection.	No standard set solely for recreational water protection.	No standard set	No standard set	Pennsylvania is currently in the process of changing recreational water quality criteria for bacteria from total coliforms to fecal coliforms standard shown on table. The fecal coliform standard is similar to that recommended by the Federal Water Pollution Control Administration.

*The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Puerto Rico	Department of Health	June, 1967	1000/100 ml as a monthly median value. Counts in excess of this shall not be present in more than 20% of the samples examined in any one month, nor exceed 2400/100 ml on any day.	No standard set	No standard set	No standard set	Non-quantitative*	Non-quantitative*	Because of the scarcity of surface water resources, and the high population density all surface waters are classified as sources of public water supply systems. Only coastal waters have a specific classification for recreational usage.
Rhode Island	Department of Health	Not given	1000/100 ml limiting value for bathing waters.	No standard set	No standard set	No standard set	No standard set	No standard set	Not given
South Carolina	Board of Health Pollution Control Authority	March, 1967	No standard set	A geometric mean of 200/100 ml nor shall more than 10% of the total samples during any 30 day period exceed 400/100 ml.	Range between 6.0-8.0, except that swamp waters may range from 5.0-8.0.	Not to exceed 93.2°F as a result of heated liquids.	Non-quantitative*	Non-quantitative*	Standards derived from previous standards and Federal Water Pollution Control Administration guidelines. Also guided by current pertinent technical publications.

*The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
South Dakota	Department of Health, Water Pollution Control Section	February 16, 1967	1000/100 ml as a monthly average; nor exceed this value in more than 20% of the samples examined in any one month nor exceed 2400/100 ml on any one day during the recreation season.	No standard set	No standard set	No standard set	No concentrations greater than 0.1 times the acute (96 hr) median lethal dose for short residual compounds, or 0.01 times the acute median lethal dose for accumulative substances or substances with residual life exceeding 30 days.	Non-quantitative	Limiting values based on literature studies including McKee and Wolf, "Water Quality Criteria," 2nd edition and U.S. Geological Survey, "Water Quality efforts for South Dakota." The criteria proposed by neighboring States were also considered.
Tennessee	Department of Public Health, Stream Pollution Control Board	May 26, 1967	No standard set	Standard for water contact recreation, not to exceed 1000/100 ml in any two consecutive samples May - September. Standard for waters used for recreation by organized camps specify - F.C. not to exceed geometric (Log) average of 200/100 ml for any five consecutive samples collected on separate days, nor shall more than 2 of any 5 consecutive samples collected on separate days exceed 1000/100 ml.	Within a range of 6.0-9.0 and shall not fluctuate more than 1.0 unit in this range over a period of 24 hours.	Shall not exceed 93°F.	Non-quantitative	Natural swimming areas of any organized camp shall have a Secchi disc clarity of 5 feet at all times.	Not given

The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Texas	Texas Water Quality Board	June, 1967	No standard set	A geometric mean of less than 200/100 ml and not more than 10% of the samples during any 30 day period should exceed 400/100 ml. This policy is advisory only and does not limit the responsibilities of local health agencies.	No standard set solely for recreational water protection.	No standard set solely for recreational water protection.	Non-quantitative*	Non-quantitative*	Values of the various parameters in the Texas water quality requirements are based on historical data, knowledge gleaned from testimony presented at 30 public hearings, professional knowledge of the Boards staff and judgement of the Board.
Utah	State Division of Health	November, 1968	50/100 ml arithmetical mean except that 20% of all samples collected in any month may exceed this standard if no more than 5% of all samples collected in the same month exceed a density of 100/100 ml.	No standard set	6.5-8.5	Waters shall be so protected against controllable pollution including heat, as to be suitable at all times for usage under the waters classification.	Non-quantitative*	Non-quantitative*	Not given
Vermont	Department of Water Resources	May 29, 1967	A median value of 500/100 ml for a minimum of five samples collected during any 30 day period, individual daily value shall not exceed 2500/100 ml.	No standard set	No reduction but allowable increase to 8.0 units.	No allowable increase except as may be consistent with fish and wildlife habitat.	Non-quantitative*	No change other than that caused by natural conditions.	[illegible]

*The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, [illegible] shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Virginia	State Water Control Board	1969	For Primary Contact Not to exceed 2,400/100 ml as a monthly average value; nor to exceed this number in more than 20% of the samples examined during any month not applicable during or immediately following periods of rainfall. For Secondary Contact Not to exceed 5,000/100 ml.	Primary Contact No standard set Secondary Contact 1,000/100 ml	No standard given	No standard given	No standard given	No standard given	Based on information available in the literature with the further philosophy that all waters would not be satisfactory for primary contact recreation and on this basis specified areas were designated for recreational use.
Virgin Islands	Office of the Commissioner of Health	January 16, 1969	No standard set	70/100 ml as a monthly median value.	7.0-8.5	Not to exceed 90°F at any time nor as a result of waste discharge to be more than 4°F above natural during fall, winter or spring nor 1.5°F above natural during summer.	Non-quantitative[1]	Non-quantitative[1]	Criteria covers coastal waters

[1] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Washington	Water Pollution Control Commission	Proposed criteria is not yet adopted.	240/100 ml with less than 20% of the samples exceeding 1000/100 ml when associated with any fecal source. The State Health Department is advocating that these figures be changed to a median value of 50 with less than 10% of the samples exceeding 230/100 ml when associated with any fecal source.	No standard set	No measurable change from natural conditions.	No measurable change from natural conditions.	Non-quantitative[1]	Turbidity shall not exceed 5 JTU over natural conditions. The State Health Department suggests that this be changed to no turbidity over natural conditions.	The State of Washington does not have a single use classification of recreation for either the intra or inter state waters. The criteria listed is proposed criteria for lakes. Basic water quality data collected over a period of years and available research pertinent to Washington State waters was used as a basis for standards
West Virginia	Department of Health	May, 1968	1000/100ml as a monthly average value, nor exceed this number in 20% of the samples examined during any month, nor exceed 2400/100 ml on any day.	No standard set	6.0-8.5	Not to exceed 87°F at any time during months of May - November, and not to exceed 73°F at any time during months of December - April.	Non-quantitative[1]	Non-quantitative[1]	Criteria covers interstate and intrastate waters classified category A for water contact recreation.

[1] The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

TABLE 41

State	Agency Responding	Date Current Standards Adopted	Bacterial		pH	Temperature Shall Not Be Increased	Pesticides and Oils	Clarity	Rationale and Remarks
			Total Coliforms Not to Exceed	Fecal Coliforms Not to Exceed					
Wisconsin	Department of Natural Resources	June, 1967	Arithmetic average of 1000/100 ml and a maximum not exceeding 2500/100 ml during the recreational season. For partial body contact - arithmetic average 5000/100 ml with no more than 1 of the samples exceeding 20,000/100 ml.	No standard set	No standard set	No standard set	No standard set	No standard set	Criteria based on available scientific knowledge and have been referred for comment to health authorities, fish and wildlife biologists and other interested professional persons. As knowledge increases the standards will be modified to reflect such increased knowledge.
Wyoming	Department of Health and Social Services, Sanitary Engineering Services	June 27, 1967	No standard set	While sample data is accumulated no individual sample shall exceed 95% confidence limit of the historical average, provided that in no case will the geometric mean of the last five consecutive samples exceed 2000/1000 ml whichever is the least.	6.5-8.5	No standard set	Non-quantitative Specific limits will not be established for toxic material as all possible compounds, combinations and effects are not known.	Non-quantitative	Bacteriological standards apply to all interstate waters, however, as a general policy, the Board of Health considers only reservoirs and lakes suitable for full body contact sports. Standards based on use criteria, existing quality and existing sources of pollutants.

The term non-quantitative refers to areas where standards are specified in phrases such as required for safety, essentially free from, and shall not be present in quantities which cause the water to be toxic to human, plant or aquatic life.

APPENDIX B

ASSUMPTIONS AND DETAILED EXAMPLES OF THE METHODOLOGY

Salmonella typhosa C_p

The summary of the raw dose response test data on human volunteers published by Hornick, et al (1966, 1970) is shown on Table 42.

To identify the probability density function which best described the universe of human capabilities from which these test subjects were drawn, it was necessary to make certain assumptions:

1. The "exact" number of S. typhosa was not measured but rather "estimated" (Hornick, 1966). For this analysis it was assumed that the number identified was the minimum of the class interval; e.g., no less than 10^3 S. typhosa were fed as the challenge dose. Just how accurate this assumption is probably will not be known until further tests are performed. This point will be discussed further in the section dealing with the R_q vector.

2. There is reasonably good and sufficient data to permit attempts to identify the universe from which the published data were sampled. Thus, the nine data points associated with the 10^8 challenge dose was given even weight with the 220 data points associated with the 10^5 challenge dose, etc.

3. It was further assumed that the universe from which the published data were sampled can be described by one of five well known probability density functions, as has already been discussed: normal, lognormal, Weibull, Gamma, or exponential. This is particularly important, because there is a paucity of data at the lower end of the dose-response capability. Thus, this low end will be obtained by extrapolation using the equation for the probability density function selected.

4. An upper limit at which certain illness would occur was assumed to be 10^{12} S. typhosa or three orders of magnitude greater than the test data for the amount of S. typhosa required to cause 98% illness.

Based on the foregoing assumptions, a histogram was constructed for the sample consisting of the published data as shown by the solid line in Figure 32. It can be seen that each step of the histogram represents an average of the data points of volunteers ill at each concentration of challenge dose.

The construction of the histogram also involves the first of the previous assumptions, that each class interval starts at the cited challange dose. This assumption is unconservative because it increased each class interval on the concentration of factor axis. An "unconservative" assumption might be to distribute the class interval equally on either side of the class mark. This

TABLE 42

S. typhosa Raw Dose Response Data*

(After Hornick, et al, 1966, 1970)

Test	*S. typhosa* Strain	Challenge Dose Concentration	Number of Volunteers Sick @ Concentration	Total Number of Volunteers Challenged @ Concentration
(a)	Quailes	10^3	0	14
		10^5	32	116
		10^7	16	32
		10^8	8	9
		10^9	40	42
(b)	Quailes	10^7	16	30
	Zermatt	10^7	6	11
	TY2V	10^7	2	6
(c)	Quailes	10^5	28	104
		10^7	15	30
		10^9	4	4

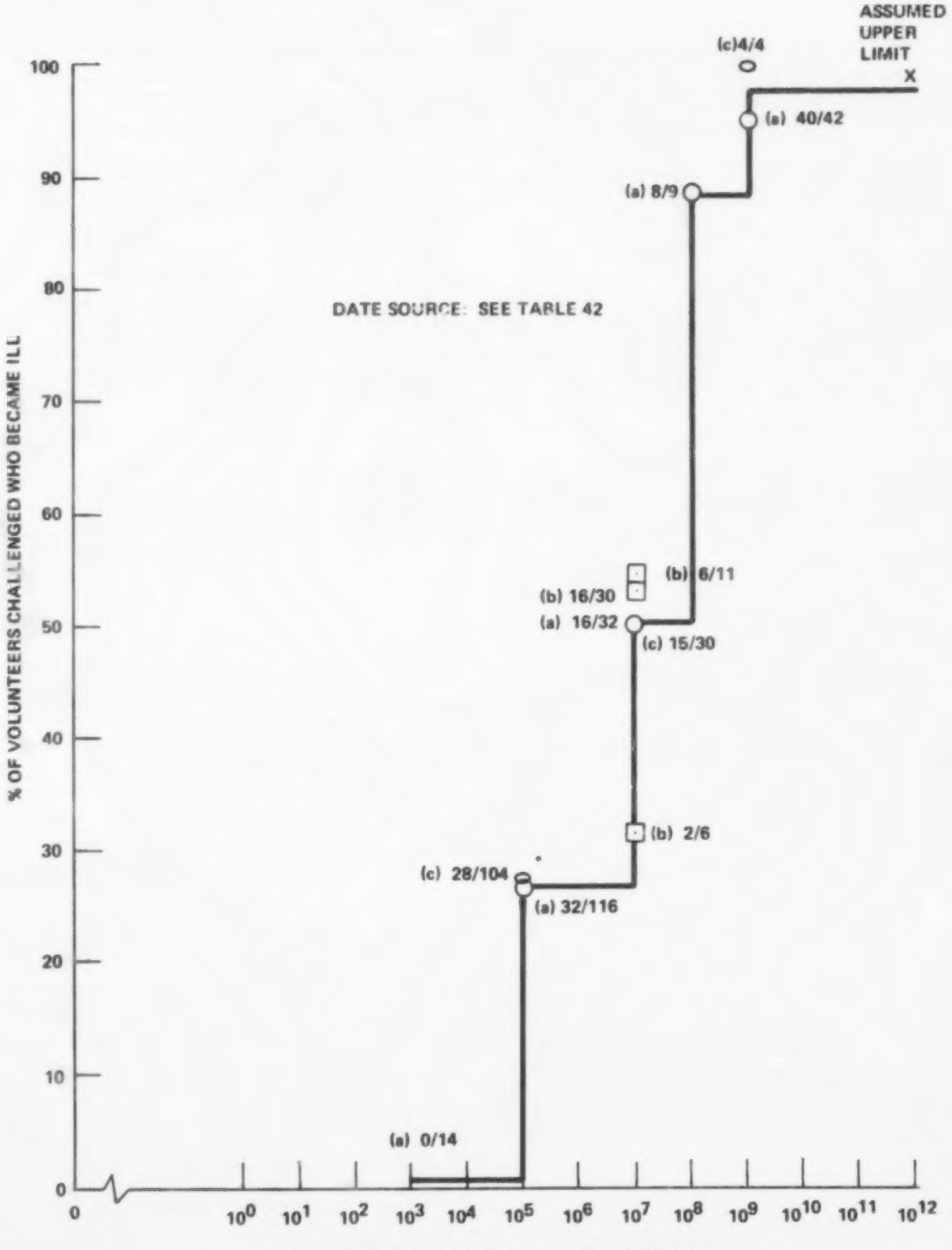

Figure 31. Histogram Constructed From Raw Data for S. typhosa

might also be an over-conservative assumption. Because of the ambiquity of the "estimation process," the entire class interval above the class mark is the same as stating that a larger concentration of challenge dose is required to make the same percent of volunteers ill. As, however, this unconservatism is removed by the definition of the requirements R_q for the *Salmonella* factor, it is discussed in that section of the report.

Twenty-five pseudo-random numbers were generated, and samples were obtained from the histogram of Figure 32. These samples were input to the BSTFIT program. The results of this analysis are shown in Table 43. This table presents the maximum likelihood estimates of the parameters describing the normal, lognormal, exponential, Weibull, and Gamma distributions. Based on the Kolmogorov-Smirnov goodness of fit (or "d") test, it can be established that the lognormal probability density function ($\hat{\sigma}_y = 3.894$, $\hat{\bar{y}} = 16.39$) is the best description of the universe from which the dose response samples was drawn. The relationship between the histogram (dash-dot line), the 25 samples drawn from it (①——①), and the lognormal distribution selected by BSTFIT is shown in Figure 33.

The curves in Figures 32 and 33 were plotted on rectilinear cartesian coordinate paper. Figure 13 in the text shows the lognormal distribution selected by BSTFIT plotted on "lognormal probability" paper. On this type plot, the density function appears as a straight line. The upper and lower 95% confidence intervals are also shown on this figure.

Salmonella - other, C_p

The *S.* - other C_p density function is based upon work published by McCullough and Eisele in 1951 dealing with strains of *S. bareilly*, *S. newport*, *S. anatum* I, II and III, *S. meleagridis* I, II and III, *S. Derby*, and *S. pullorum* I, II, III and IV. Thus groups of the Kauffman-White Scheme B, C_1, C_2, D, and E, the majority of the groups significant to humans, were covered. As in the case of *S. typhosa*, the pathogens were fed to the volunteers; thus the results are directly applicable to the case of recreation water quality. These tests were performed in a way to emphasize the detection of a "minimum effective dose." They were conducted in a manner quite similar to "step stress testing to malfunction." Thus the dose response data could be input to BSTFIT directly without recourse to sampling from a histogram.

The input to BSTFIT is shown in Table 44. The results are shown in Table 45. once again the lognormal probability density function has been selected ($\hat{\bar{y}} = 17.86$, $\hat{\sigma}_y = 3.698$) at the 5% level of significance. This probability density function is shown graphically in Figure 14 in the text, along with its upper and lower 95% confidence limits.

TABLE 43

Salmonella typhosa Capabilities Vector (C_p) Using 25 Random Numbers

Name of Distribution	Density Function	Maximum Likelihood Estimation of Parameters	Iteration	"d" Statistic	Probability that data came from Cited Distribution
Normal	$f(x) = \frac{1}{\sigma\sqrt{2\pi}} e - \frac{1}{2}\left(\frac{x-\bar{x}}{\sigma}\right)^2$	$\hat{\bar{x}} = 4.011 \times 10^{10}$ $\hat{\sigma} = 2.000 \times 10^{11}$	---	0.538	0 +
Lognormal	$f(x) = \frac{1}{cy\sqrt{2\pi}} e - \frac{1}{2}\left(\frac{\log x-\bar{y}}{\sigma_y}\right)^2$ where $y = \log x$	$\hat{\bar{y}} = 16.39$ $\hat{\sigma}_y = 3.894$ (best distribution)	---	0.191	0.317
Exponential	$f(x) = \theta e^{-\theta x}$	$\hat{\theta} = 2.393 \times 10^{-11}$	---	0.936	0 +
Gamma	$f(x) = \frac{1}{\theta\Gamma(\alpha)} \left[\frac{x-c}{\theta}\right]^{\alpha-1} \exp\left[\frac{-(x-c)}{\theta}\right]$	$\hat{\theta} = 6.066 \times 10^{11}$ $\hat{\alpha} = 0.06613$ $\hat{c} = 1.0 \times 10^5$	29	0.2981	0.0235
Weibull	$f(x) = \theta_1 \theta_2 x^{(\theta_1 - 1)} \exp\left[-\theta_2 x^{\theta_1}\right]$	No reasonable fit possible	---	---	---

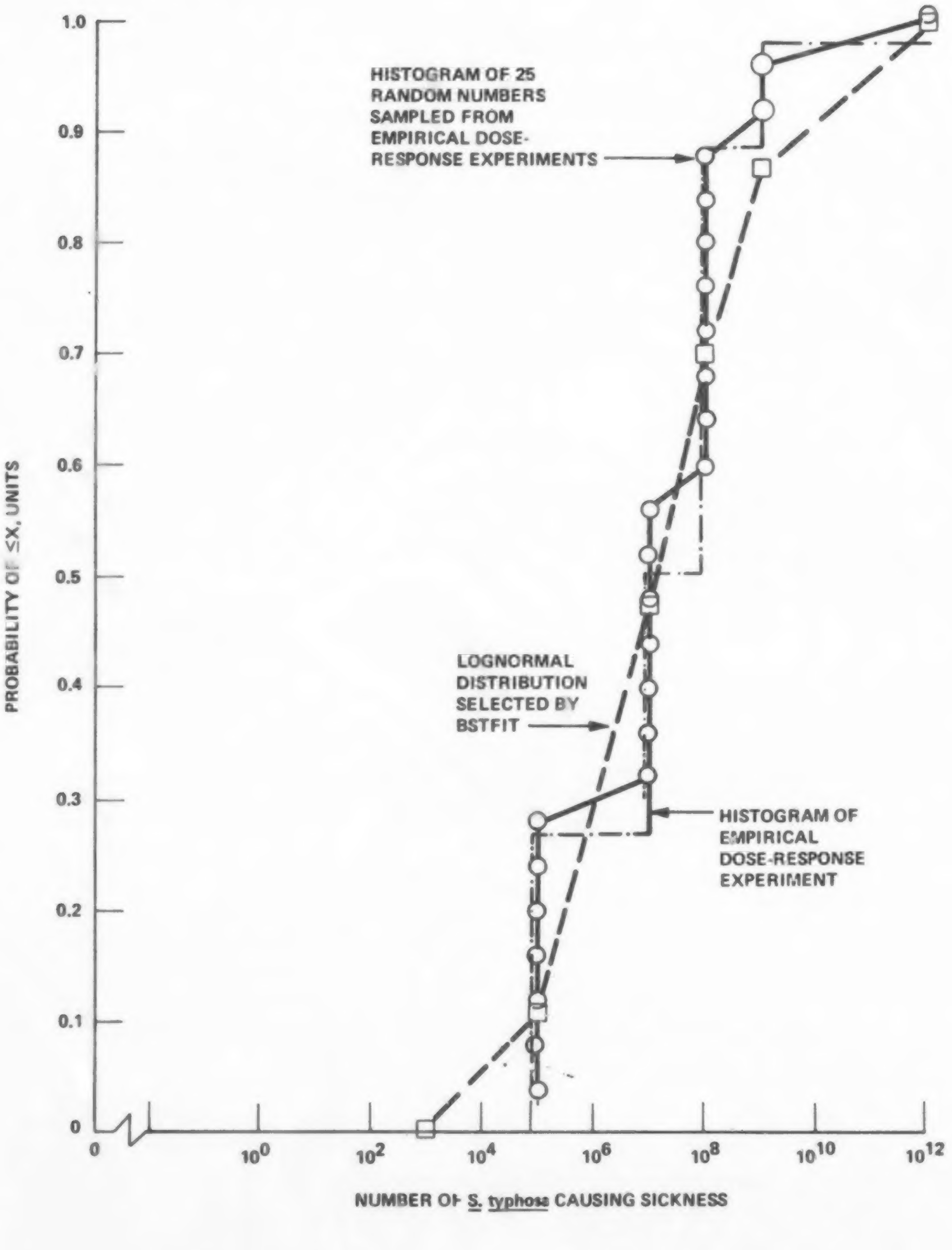

Figure 32. S. typhosa C_p As Selected By BSTFIT

TABLE 44

Summary of *Salmonella*-other Dose
Response Data Input to BSTFIT
(After McCullough and Eisele, 1951)

Concentration of Challenge Dose	Strain	No. of Volunteers Ill/group of 6
0.125 x 10^6	*S. bareilly*	1
0.152 x "	*S. newport*	1
0.385 x "	*S. newport*	1
0.587 x "	*S. anatum* I	2
0.695 x "	*S. bareilly*	2
0.860 x "	*S. anatum* I	3
1.275 x "	*S. anatum* III	2
1.350 x "	*S. newport*	3
1.70 x "	*S. bareilly*	4
4.675 x "	*S. anatum* III	4
7.675 x "	*S. meleagridis* III	1
10.0 x "	*S. meleagridis* II	1
10.0 x "	*S. meleagridis* III	2
15.0 x "	*S. derby*	3
20.0 x "	*S. meleagridis* II	2
24.0 x "	*S. meleagridis* I	1
41.0 x "	*S. meleagridis* II	5
44.5 x "	*S. anatum* II	1
50.0 x "	*S. meleagridis* I	4
67.25 x "	*S. anatum* II	4
1.280 x 10^9	*S. pullorum* IV	3
3.975 x "	*S. pullorum* IV	2
6.750 x "	*S. pullorum* II	4
7.640 x "	*S. pullorum* III	1
10.0 x "	*S. pullorum* I	6
16.0 x "	*S. pullorum* I	6

TABLE 45

Salmonella Total Requirements Vector (R_q) Using 25 Random Numbers

Name of Distribution	Density Function	Maximum Likelihood Estimation of Parameters	Iteration	"d" Statistic	Probability that data came from Cited Distribution
Normal	See TABLE 43	Not a reasonable fit	---	---	---
Lognormal	See TABLE 43	$\hat{y} = -2.076$ $\hat{\sigma}_y = 6.198$	---	0.364	0.00265
Exponential	See TABLE 43	$\hat{\theta} = .0774$	---	0.360	0.00307
Gamma	See TABLE 43	$\hat{\theta} = 22.90$ $\hat{\alpha} = 0.568$ $\hat{c} = 0$	27	0.359	0.00317
Weibull	See TABLE 43	$\theta_1 = 0.599$ $\theta_2 = 0.283$ (preferred distr.)	---	0.359	0.00319

SECTION XV

BIBLIOGRAPHY

A. GENERAL BIBLIOGRAPHY

BIBLIOGRAPHY

1. Allen, L. A., J. Grindley, and E. Brooks, "Some Chemical and Bacterial Characteristics of Bottom Deposits from Lakes and Estuaries," J. Hyg. (Camb.), 51:185-192 (1953).

2. Allen, L. A., M.A.F. Pierce, and H. M. Smith, "Enumeration of Streptococcus faecalis, with particular reference to polluted waters," J. Hyg. (Camb.), 51:458-465 (1953).

3. Allen, L. A., S. M. Pasley, and M.A.F. Pierce, "Some Factors Affecting the Viability of Fecal Bacteria in Water," J. Gen. Microbiol., 7:36-46 (1952).

4. von Alvin, W. H. (1946), Ed. Reliability Engineering, pp 171-172, Prentice Hall Book Co., New Jersey (1970).

5. American Public Health Association, Report of the Committee on Bathing Places, Am. J. Pub. Health, 12:121-3 (1922).

6. American Public Health Association, Report of the Joint Committee Bathing Places of the A.P.H.A. and the Conference of State Sanitary Engineers, Am. J. Pub. Health, 23:40-49 Year Book (1933).

7. American Public Health Association, Progress report of the Joint Committee on Bathing Places of the Conference of State Sanitary Engineers and the Public Health Engineering Section of the A.P.H.A., Am. J. Pub. Health, 26:209-220, Year Book (1936).

8. American Public Health Association, Progress report of the Joint Committee on Bathing Places, Am. J. Pub. Health, 30:50-51 Year Book (1940).

9. American Public Health Association, Recommended Practice for the Design, Equipment, and Operation of Swimming Pools and Other Public Bathing Places, New York, A.P.H.A. (1949).

10. American Public Health Association, Ibid, 10th ed., (1957).

11. American Public Health Association, Standard Methods for the Examination of Water and Wastewater, A.P.H.A. 12th ed., New York (1965).

12. American Society of Civil Engineers Public Health Activities Committee "Coliform Standards for Recreational Waters," J. Sanit. Eng. Div. SA-4 (1963), SA-1 (1964), SA-2(1964), SA-5 (1964).

13. American Society of Civil Engineers "Engineering Evaluations of Virus Hazard in Water," J. Sanit. Eng. Div. SA-1, 111-161, (1970).

14. American Water Works Association, "Viruses in Water, Committee Report," J. A.W.W.A. 61, 491-494 (1969).

15. Anderson, E. S., N. S. Galbraith, and C. E. D. Taylor, "An Outbreak of Human Infection due to *Salmonella typhimurium*, phage-type 20a, Associated with Infection in Calves," Lancet, i:854-858 (1961).

16. Anderson, J. R., "A Study of the Influence of Cyanuric Acid on the Bactericidal Effectiveness of Chlorine," Am. J. Pub. Health, 55:1629-1637 (1965).

17. Andre, D. A., H. H. Weiser, and G. W. Melaney, "Survival of Bacterial Enteric Pathogens in Farm Pond Water," J. Am. Water Work Assoc., 59:503-508 (1967).

18. Andrewes, C. H., and A. C. Allison, "Newcastle Disease as a Model for Studies of Experimental Epidemiology," J. Hyg., Camb., 59:285-293 (1961).

19. Anon., "Typhoid Fever in Minneapolis and Gastroenteritis in Milwaukee," J. Am. Water Works Assoc., 31:374-383 (1939).

20. Anon., "An Investigation of the Efficacy of Submarine Outfall Disposal of Sewage and Sludge," California State Water Poll. Cont. Bd., Pub. No. 14, Sacramento (1956).

21. Anon., "Carbon Chloroform Extract (CCE) in Water, "Subcommittee on Organic Chemicals of AWWA Committee 8930P., J. Am. Water Works Assoc., 54:223-227 (1962).

22. Anon., "Microbiological Content of Domestic Waste Waters Used for Recreational Purposes," California Water Quality Control Board, Pub. No. 32, 50 pp., Sacramento (1965).

23. Armstrong, R. W., "Environmental Factors Involved in Studying the Relationship Between Soil Elements and Disease," Amer. J. Pub. Health, 54:1536-1544 (1964).

24. A.S.C.E., "Coliform Standards for Recreational Waters," Progress Report of the Public Health Activities Committee, Sanitary Engineering Division, J. San. Eng. Div., Proc. Am. Soc. Civil Eng., 89:SA4, 57-94, (1963).

25. Askew, J. B., R. F. Bott, R. E. Leach, and B. L. England, "Microbiology of Reclaimed Water from Sewage for Recreational Use at Santee, California," Am. J. Pub. Health, 55:453-462 (1965).

26. Bartley, C. H., and L. W. Slanetz, "Types and Sanitary Significance of Fecal Streptococci Isolated from Feces, Sewage and Water," Am. J. Pub. Health, 50:1545-1552 (1960).

27. Beard, J. W., "Host-virus Interaction in the Initiation of Infection," In *Transmission of Viruses by the Water Route* (edited by G. Berg), John Wiley & Sons, New York, p. 167-192 (1967).

28. Beard, P. J., "The Survival of Typhoid in Nature," J. Am. Water Works Assoc., 30:124-131 (1938).

29. Beard, P. J., and N. F. Meadowcroft, "Survival and Rate of Death of Intestinal Bacteria in Sea Water," Am. J. Pub. Health, 25:1023-1026 (1935).

30. Barbaro, R. D. Carroll, B. J., Tebo, L. B. and Walters, L. C., "Bacteriological Water Quality of General Recreational Areas in the Ross Barnett Reservoir," J. WPCF 41, 1330-1339 (1969).

31. Beeton, A. M., "Relationship Between Secchi Disc Readings and Light Penetration in Lake Huron," Trans. Am. Fish Soc., 87:73-79 (1958).

32. Begbie, R. S., and H. J. Gibson, "Occurrence of Typhoid-Paratyphoid Bacilli in Sewage," Brit. Med. J., 2:55-56 (1930).

33. Belding, H. S. and Hatch, T. F., "Index for Evaluating Heat Stress in Terms of Resulting Physiological Strains," Am. Soc. Htg. & Air Condition. Engrs., Transactions 62, 213-236 (1956).

34. Bell, J. A., W. P. Rowe, J. L. Engler, R. H. Parrott, and R. J. Huebner, "Pharyngo-Conjunctival Fever:Epidemiological Studies of a Recently Recognized Disease Entity," J. Am. Med. Assoc., 157:1083-1092 (1955).

35. Berg, G., "The Virus Hazard in Water Supplies," J. New Eng. Water Works Assoc., 78:79-104 (1964).

36. Berg, G., "Die Virusubertragung auf dem Wasserweg," Arch. fur Hyg. und Bakter., 149:310-335 (1965) W65-28.

37. Berg, G., "Virus Transmission by the Water Vehicle, I. Viruses," Health Lab. Sci., 3:86-89 (1966).

38. Berg, G., "Virus Transmission by the Water Vehicle, II. Virus Removal by Sewage Treatment Procedures," Health Lab. Sci., 3:90-100 (1966).

39. Berg, G., "Virus Transmission by the Water Vehicle, III. Removal of Viruses by Water Treatment Procedures," Health Lab. Sci., 3:170-181 (1966).

40. Berg, G., Transmission of Viruses by the Water Route, John Wiley & Sons, New York, 289 pp. (1967).

41. Berg, G., S. L. Chang, and E. K. Harris, "Devitalization of Microorganisms by Iodine," Virology, 22:469 (1964).

42. Berg, G., R. B. Dean, and D. R. Dahling, "Removal of Poliovirus I. from Secondary Effluents by Lime Flocculation and Rapid Sand Filtration." J. Am. Water Works Assoc., 60:193-198 (1968).

43. Berg, G., D. Berman, S. L. Chang, and N. A. Clarke, "A Sensitive Quantitative Method for Detecting Small Quantities of Virus in Large Volumes of Water," Am. J. Epidemiol., 83:196-203 (1966).

44. Berg, G., G. Stern, D. Berman, and N. A. Clarke, "Stabilization of Chemical Oxygen Demand in Primary Wastewater Effluents by Inhibition of Microbial Growth," J. Water Poll. Cont. Fed., 38:1472-83 (1966).

45. Bernstein, I. L., and R. S. Sofferman, "Sensitivity of Skin and Bronchial Mucosa to Green Algae," J. Allergy, 38:166-173 (1966).

46. Berry, A. E., and A. V. Delaporte, "Standards and Regulations for Quality of Water in Bathing Places," J. Am. Water Works Assoc., 33:195-200 (1941).

47. Black, A. P. and S. A. Hannah, "Measurement of Low Turbidities," J. Amer. Water Works Assoc., 57:901-916 (1965).

48. Booth, R. L., J. N. English, and G. N. McDermott, "Evaluation of Sampling Conditions in the Carbon Adsorption Method," J. Amer. Water Works Assoc., 57:215-220 (1965).

49. Borneff, J., "Cancerogene Substanzen in Wasser und Boden, IV., Arch. fur Hyg. und Bakteriol., 144:249-262 (1960).

50. Borneff, J. and R. Fischer, "Cancerogene Substanzen in Wasser und Boden, VIII. Untersuchungen an Filter-Aktiokahle Nach Verwendung im Wasserwerks," Arch. fur Hyg. und Bakteriol., 146:14-16 (1962).

51. Borneff, J. and R. Knerr, "Cancerogene Substanzen in Wasser und Boden, III. Quantitative Ermittlungen zur Laslichkeit, Filtration, Adsorption und Eindringtiefe," Arch. fur Hyg. und Bakteriol., 144:81-93 (1960).

52. Breed, R. S., E. G. D. Murray, and N. R. Smith, *Bergey's Manual of Determinative Bacteriology*, 7th ed., The Williams and Wilkins Co., (1957).

53. Brezenski, F. T., R. Russomano, and P. Defalco, Jr., "The Occurrence of *Salmonella* and *Shigella* in Post-chlorinated and Non-chlorinated Sewage Effluents and Receiving Waters," Health Lab. Sci., 2:40-46 (1965).

54. Broek, J. C. H., and C. P. Mom, "Contamination of Polder Water with *Salmonella paratyphi* as a Consequence of the Discharge of Sewage," Leeuenhoek ned. Tÿdschr., 19:363 (1953); Water Poll. Abst., 27:1514 (1954).

55. Browning, G. E., and J. O. Mankin, "Gastroenteritis Epidemic Owing to Sewage Contamination of Public Water Supply," J. Am. Water Works Assoc., 58:1465-1470 (1966).

56. Bruch, H. A., W. Ascoli, N. S. Scrimshaw, and J. E. Gordon, "Studies of Diarrheal Disease in Central America. V. Environmental Factors in the Origin and Transmission of Acute Diarrheal Disease in Four Guatemalan Villages," Am. J. Trop. Med. Hyg., 12:567-579 (1963).

57. Brunner, G., "Fish as Carriers of Bacteria," Vom Wasser (Ger.) 1949, 17:9-20, J. Am. Water Works Assoc., 44:82 (1952, abst.).

58. Buczowska, Z., "Research on the Bacteriological Pollution of Coastal Waters," Bull. Inst. Marine Med. Gdansk., 10:141-150, Bull. Hyg. (1960), 35:426 (1959).

59. Buczowska, Z., "Tubercle Bacilli in the Sewage and in Sewage-Receiving Waters, Bull. Inst. Marine Med. in Gdansk., 16:49-56 (1965).

60. Bureau of Outdoor Recreation, Outdoor Recreation Trends, Supt. of Documents, U. S. Govt. Printing Office, Washington, D. C. (1967).

61. Burns, C., "Infectious Hepatitis: Its Incidence in Leister and General Epidemiology," J. Roy. Soc. Health, 85:144-148 (1965).

62. Bursche, E. M., "Beiträge zur Frage des 'Krautschwundes' in H_2S-Oscillatorien-Seen," Zeit. Fisch., 4:53-99 (1955). (Cited by Mackenthun and Ingram, 1967).

63. Butterfield, C. T., "Observations on Changes in Numbers of Bacteria in Polluted Water," Sewage Works J., 5:600-615 (1933).

64. Butterfield, C. T., and E. Wattie, "Influence of pH and Temperature on the Survival of Coliform and Enteric Pathogens When Exposed to Chloramine," Pub. Hlth. Rep., 61:157-192 (1946).

65. Butterfield, C. T., E. Wattie, S. Megregian, and C. W. Chambers, "Influence of pH and Temperature on the Survival of Coliforms and Enteric Pathogens When Exposed to Free Chlorine," Pub. Hlth. Rep., 58:1837-1866 (1943).

66. Byrd, O. E., H. M. Malkin, G. B. Reed, and H. W. Wilson, "Safety of Iodine as a Disinfectant in Swimming Pools," Pub. Hlth. Rep., 78:393-397 (1963).

67. Champ, T. R., Water and Its Impurities, Reinhold Pub. Corp., New York, 355 pp., (1963).

68. Carlson, G. F., Jr., F. E. Woodard, D. F. Wentworth, and O. J. Sprone, "Virus Inactivation on Clay Particles in Natural Waters," J. Water Poll. Cont. Fed., 40:R89-106 (1968).

69. Carlucci, A. R., and D. Pramer, "Factors Affecting the Survival of Bacteria in Sea Water," Appl. Microbiol., 7:388-92 (1959).

70. Carswell, J. K., J. M. Symons, and G. C. Robeck, "Research on Recreational Use of Watersheds and Reservoirs," J. Am. Water Works Assoc., 61:297-304 (1969).

71. Cate, T. R., R. B. Couch, and K. M. Johnson, "Studies with Rhinoviruses in Volunteers: Production of Illness, Effect of Naturally Acquired Antibody, and Demonstration of a Protective Effect Not Associated with Serum Antibody," J. Clinical Investigation 43, 56-67 (1964).

72. Cate, T. R., R. B. Couch, W. F. Fleet, W. R. Griffith, P. J. Gerone, and V. Knight, "Production of Tracheobronchitis in Volunteers with Rhinovirus in a Small-Particle Aerosol," Am. J. Epidem. 81, 95-105 (1965).

73. Chambers, C. W. and N. A. Clarke, *Health Aspects of Poultry Waste Disposal*, Proc. 2nd, Nat. Symposium on Poultry Waste Management, Lincoln, Nebraska, pp. 193-212 (1964).

74. Chang, S. L., "Viruses, Amebas, and Nematodes in Public Water Supplies," J. Amer. Water Works Assoc., 53:288-296 (1961).

75. Chang, S. L., "Waterborne Viral Infections and Their Prevention," Bull. Wld. Hlth. Org., 38:401-414 (1968).

76. Change, S. L., and P. W. Kabler, "Free-Living Nematodes in Aerobic Treatment Plant Effluent," J. Water Poll. Cont. Fed., 34:1256-61 (1962).

77. Chang, S. L., G. Berg, N. A. Clarke, and P. W. Kabler, "Survival, and Protection Against Chlorination, of Human Enteric Pathogens in Free-living Nematodes Isolated from Water Supplies," Am. J. Trop. Med. Hyg., 9:136-142 (1960).

78. Chang, S. L., R. E. Stevenson, A. R. Bryant, R. L. Woodward, and P. W. Kabler, "Removal of Coxsackie and Bacterial Viruses in Water by Flocculation," Am. J. Pub. Hlth., 48:51-61; 159-169 (1958).

79. Chase, F. E., and M. L. Wright, "*Salmonella* in Dried Eggs," Food Ind., 18:968 (1946). See also: "The Occurrence and Distribution of *Salmonella* Types in Fowl. I. Isolation from Hen's Eggs," Can. J. Res., 24F:77-80 (1946).

80. Chernukka, Y. G., and E. V. Karaseva, "Leptospira Infection of Serotype Lora (Australis Group) found in the U.S.S.R.," Trop. Geogr. Med., 1:22-25 (1965).

81. Chin, T. D. Y., W. H. Mosley, S. Robinson, and C. R. Gravelle, "Detection of Enteric Viruses in Sewage and Water," in Transmission of Viruses by the Water Route, G. Berg., ed., John Wiley and Sons, New York, pp. 389-400 (1967).

82. Clark, H. S., and P. W. Kabler, "Reevaluation of the Significance of the Coliform Bacteria," J. Amer. Water Works Assoc., 56:931-936 (1964).

83. Clark, H. F., P. W. Kabler, and E. E. Geldreich, "Advantages and Limitations of the Membrane Filter Procedure," Water and Sewage Works, 104:385-387 (1957).

84. Clark, H. F., E. E. Geldreich, H. L. Jeter, and P. W. Kabler, "The Membrane Filter in Sanitary Bacteriology," Pub. Hlth. Rep., 66:951-977 (1951).

85. Clark, H. F., E. E. Geldreich, P. W. Kabler, R. H. Bordner, and C. B. Huff, "The Coliform Group. I. Boric Acid-Lactose Broth Reactions of Coliform IMViC types," Appl. Microbiol., 5:396-420 (1957).

86. Clark, R. N., "The Transmission of Disease by Sewage," Sewage Works J., 18:1139-1146 (1946).

87. Clarke, G. L., "The Utilization of Solar Energy by Aquatic Organisms," Prob. Lake Biology, A.A.A.S. Publ., 10:27-38 (1939).

88. Clarke, N. A., and S. L. Chang, "Enteric Viruses in Water," J. Am. Water Works Assoc., 51:1299-1317 (1959).

89. Clarke, N. A. and P. W. Kabler, "Human Enteric Viruses in Sewage," Health Lb. Sci., 1:44-50 (1964).

90. Clarke, N. A., R. E. Steventson, and P. W. Kabler, "Survival of Coxsackie Virus in Water and Sewage," J. Am. Water Works Assoc., 48:677-682 (1956).

91. Clarke, N. A., et al, "Human Enteric Viruses: Source, Survival, and Removability in Waste Water," J. Water Poll. Cont. Fed., 34:249-255 (1962).

92. Clarke, N. A., G. Berg, P. W. Kabler, and S. L. Chang, "Human Enteric Viruses in Water: Source, Survivability, and Removability," In W. W. Eckenfelder, ed., Advances in Water Pollution Research, Proc. Int. Conf. London, September, 1962, Vol. 2, Pergamon, London, p. 523-536 (1964).

93. Clarke, N. A., R. E. Stevenson, S. L. Chang, and P. W. Kabler, "Removal of Enteric Viruses from Sewage by Activated Sludge Treatment," Amer. J. Pub. Hlth., 51:1118-1129 (1961).

94. Coetze, O. J., "Comments on Sewage Contamination of Coastal Bathing Waters," South African Med. J., 35:261-269 (1961).

95. Collet, E., R. Johnston, L. F. Ey, and C. C. Croft, "Isolation of Salmonella typhosa from Well Water by the Membrane Filter Technic," Am. J. Pub. Health, 43:1438 (1953).

96. Comley, H. H., "Cyanosis in Infants Caused by Nitrates in Well Water," J. Amer. Med. Assoc., 129:112-116 (1945).

97. Cooke, W. B., "Fungi in Polluted Water and Sewage," Sew. Ind. Wastes, 26:539-49, 661-74, 790-94 (1954).

98. Cottam, C., "Pesticides and Water Pollution," Proc. Nat. Conf. on Water Poll., Washington, D. C., p. 222 (1960).

99. Cox, C. R., "Waterborne Gastroenteritis," J. Am. Water Works Assoc., 31:489-501 (1939).

100. Cox, C. R., "Acceptable Standards for Natural Waters Used for Bathing," Proc. Am. Soc. Civil Engrs., 77:Sep. 74 (1951).

101. Cruickshank, R., "Diarrheal Disease," Milbank Mem. Fund Quart. Suppl., 43:215-7, 240-57 (1965).

102. Cuthbert, W. A., J. J. Panes, and E. C. Hill, "Survival of Bacterium coli Type I and Streptococcus faecalis in Soil," J. Appl. Bacteriol., 18:408; Bull. of Hyg., 31:552 (1955 and 1956, respectively).

103. Dale, W. E., and G. E. Quinby, "Chlorinated Insecticides in the Body Fat of People in the U.S.," Science, 142:593-595 (1963).

104. Dambach, C. A., "Recreational Use of Impounding Reservoirs," J. Am. Water Works Assoc., 48:517-524 (1956).

105. Dammin, G. J., "The Pathogenesis of Acute Diarrheal Disease in Early Life.," Bull. Wld. Hlth. Org., 31:29-32 (1964).

106. Darasse, H., L. Le Minor, and M. Lecomte, "Isolement de Plusiers Salmonella dans une Eau de Distribution," Bull. Soc. Pathol. Exot., 52:53 (1959).

107. Dauer, C. C., "1951 Summary: Food and Waterborne Disease Outbreaks," Pub. Hlth. Rep., 67:1089-1098 (1952).

108. Dauer, C. C., "1960 Summary of Disease Outbreaks and a 10-Year Resume," Pub. Hlth. Rep., 76:915-922 (1961).

109. Dauer, C. C., and D. J. Davids, "1958 Summary of Disease Outbreaks," Pub. Hlth. Rep., 74:715-721 (1959).

110. Dauer, C. C., and G. Sylvester, "1953 Summary of Disease Outbreaks," Pub. Hlth. Rep. 69:538-546 (1954).

111. Davis, B. D., R. Dulbecca, H. N. Eisen, H. S. Ginsberg, and Wood, W. B., Jr., Principals of Microbiology and Immunology, pp 661-671, Harper and Row, New York (1968).

112. Deaner, D. G. and K. D. Kerri, "Regrowth of Fecal Coliforms," J. Am. Water Works Assoc., 465-468 (1969).

113. de Capito, T., "Isolation of Salmonella from Flies," Amer. J. Trop. Med. Hyg., 12:892-903 (1963).

114. Dedie, K., "Organisms in Sewage Pathogenic to Animals," Stadt Hygiene, 6:177-185 (1955).

115. Delos, J. S., "Bacterial Survey of Streams and Bathing Beaches at Cleveland," Sew. Ind. Wastes, 22:1618-1625 (1950).

116. Denecke, K., "Investigations on the Detection of Pathogenic Bacteria in Irrigation with Sewage," Arch. Hyg., Berl., 141:624 (1957).

117. Dennis, J. M., and A. Wolman, "1955-56 Infectious Hepatitis Epidemic in Delhi, India" J. Am. Water Works Assoc., 51: 1288-1295 (1959).

118. Dias, F. F., "Studies on the Bacteriology of Sewage," J. Indian Inst. of Sci., 45:36-48 (1963).

119. Dias, F. F. and J. V. Bhat, "Microbial Ecology of Activated Sludge. I. Dominant Bacteria," Appl. Microciol., 12:412-417 (1964).

120. Dias, F. F. and J. V. Bhat, "Microbial Ecology of Activated Sludge. II. Bacteriophages, Bdellovibrio, Coliforms, and Other Organisms." Appl. Microbiol., 13:257-261 (1965).

121. Ditton, R. B., "The Identification and Critical Analysis of Selected Literature Dealing with the Recreational Aspects of Water Resources Use, Planning and Development," Research Report No. 23, Univ. of Illinois Water Resources Center, Urbana (Research Report to Office of Water Resources Research, U.S. Dept. of Interior) (1969).

122. Dixon, F. R., and L. J. McCabe, "Health Aspects of Wastewater Treatment," J. Water Poll. Cont. Fed., 36:984-989 (1964).

123. Dubois, T., "Pollution of Swimming Bath Water, Epidemiological Aspects and Purification Problems," Arch. Belges. Med. Soc., 13:1; Bull. Hyg., London 30:790 (1955); Water Poll. Abs., 28:2633, (1955).

124. Dudley, R. H., The Economics of Reliability, Dissertation, Univ. of Southern Calif., Los Angeles (1970).

125. Duggan, R. E., and G. Q. Lipscomb, "Dietary Intake of Pesticide Chemicals in the United States (II), June 1966-April 1968," Pest. Monit. J., 2:153-162 (1969).

126. Dunlop, S. G., "The Survival of Pathogenic Organisms in Sewage," Public Works, 88:80-81 (1957).

127. Dunlop, S. G., R. M. Twedt, and W. L. Wang, "Salmonellae in Irrigation Waters," Sewage and Ind. Wastes, 23:1118-1122 (1951).

128. Dunlop, S. G., R. M. Twedt, and W. L. Wang, "Quantitative Estimation of Salmonella in Irrigation Waters," Sewage and Ind. Wastes, 24:1015-1020 (1952).

129. Dunsmore, H. J., "Criteria for Evaluation of Environmental Health Progress," Amer. J. Pub. Hlth., 54:7-10 (1964).

130. Durham, W. F., "Pesticide Residues in Foods in Relation to Human Health," Residue Reviews, 4:34-81 (1963).

131. Duuren, F. A. von, "Removal of Microorganisms from Water. I. Introduction, Water-borne Disease and the Microorganisms Involved," Water and Wastes Engr., 71:321-335; (1967); Water Poll. Abst. (Brit.) 41:1157 (1968).

132. Dyadichev, N. R., "The Survival and Multiplication of Typhoid, Paratyphoid and Dysenteric Bacteria in Water," Hygiene and San. (USSR), 24:11 (1959) (ex McKee, 1963).

133. Eaton, R. D. P., "An Outbreak of Infectious Hepatitis," Can. J. Pub. Hlth., 52:297-303 (1961).

134. Eaves, G. N. and J. O. Mundt, "Distribution and Characterization of Streptococci from Insects," J. Insect. Pathol., 2:289-298 (1960).

135. Edwards, P. R.,"Salmonella and Salmonellosis," Ann. N. Y. Acad. Sci., 66:44-53 (1956).

136. Edwards, P. R., "Salmonellosis: Observations on Incidence and Control," Ann. N. Y. Acad. Sci., 70:598-613 (1958).

137. Edwards, P. R., and W. H. Ewing, Identification of Enterobacteriaceae, Burgess Pub. Co., Minneapolis, 425 pp. (1962).

138. Eichna, L. W., and Shelley, W. B., "The Upper Limits of Environmental Heat and Humidity Tolerated By Acclimatized Men Working in Hot Environments, "J. Ind. Hyg. Toxicol. 27, 59-84 (1945).

139. Eliassen, R., "Challenges in Research and Development," J. Water Poll. Cont. Fed., 35:267-274 (1963).

140. Eliassen, R., and R. H. Cummings, "Analysis of Water-borne Outbreaks," 1938-45, J. Am. Water Works Assoc., 40:509-515 (1948).

141. Emanuel, M. L., I. M. Mackerras, and D. J. W. Smith, "The Epidemiology of Leptospirosis in North Queensland," J. Hyg., 62:451-484 (1964).

142. Emili, H., and P. S. Tomasic, "The Health Aspects of Polluted Water with Special Reference to the Epidemiology of Water-borne Infections," Water Pollution in Europe, Fourth European Seminar for Sanitary Engineers, 22:47-69 (1954).

143. England, B., R. E. Leach, B. Adame, and R. Shiosaki, "Virologic Assessment of Sewage Treatment at Santee, California. In Transmission of Viruses by the Water Route, John Wiley & Sons, New York, pp. 401-417 (1967).

144. Ettinger, M. B., "Proposed Toxicity Screening Procedure for Use in Protecting Drinking-water Quality," J. Amer. Water Works Assoc., 52:689-694 (1960).

145. Ettinger, M. B., "Sociological and Technological Factors in Developing Water Quality Criteria," Pub. Health Rep., 78:398-402 (1963).

146. Evans, F. L., III, E. E. Geldreich, S. R. Weibel, and G. G. Robeck, "Treatment of Urban Storm Water Runoff," J. Water Poll. Cont. Fed., 40:R162-170 (1968).

147. Fair, G. M., and J. C. Geyer, "Elements of Water Supply and Waste-Water Disposal, John Wiley & Sons, New York (1965).

148. Falls, J. A. (ed.), Exercise Physiology, Academic Press, New York, 471 pp. (1968).

149. Faulkner, J. A., "Physiology of Swimming and Diving." In Falls, J. A., Exercise Physiology, Academic Press, New York, pp. 415-446 (1968).

150. Faust, S. D., "Pollution of the Water Environment by Organic Pesticides," Clin. Pharm. Ther., 5:677-686 (1964).

151. Feigin, R. D., "Salmonella Everywhere, Epidemologic Diagnostic and Therapeutic Dilemmas," Clinical Pediatrics, 9:527-533.

152. Ferramola, R., R. A. Leiguardia, E. M. Ansiaume, O. A. Peso, and A. Z. R. Palazzolo, "Study of the Contamination of Surface Waters by Bacteria of the genus Salmonella," Rev. abr. Sanit. Nac., B. Aires, 33:94 (1954).

153. Flynn, M. J. and Thistlethwayte, D. K. B., "Sewage Pollution and Sea Bathing" Int. J. Water Poll. 9:641-653 (1965).

154. Foliquet, J. M., and L. Schwartzbrod, "Research on Viruses Found in Sewage and Drinking Water in Meurthe and Moselle. I. Purposes, Methods and Preliminary Results, Rev. Hyg. et Med. Soc., 13:137-162 (1965).

155. Foster, D. H., "Factors Influencing Bathing Water Quality," Thesis, Tufts University (1968).

156. Fraser, M. H., W. B. Reid and J. F. Malcolm, "The Occurrence of Coli- erogenes Organisms on Plants," J. Appl. Bacteriol., 19:301-309 (1956).

157. Freitag, J. L., "Outbreak of Dysentery," Health News, 37:4-10 (1960).

158. Gaines, S., U. Achavasmith, M. Thareesawat, and C. Duangmani, "Types and Distribution of Enteropathogenic Escherichia Coli in Bangkok, Thailand, "Amer, J. Hyg., 80:388-394 (1964).

159. Galbraith, N. S., "Pet Foods and Garden Fertilizers as Sources of Human Salmonellosis," Lancet, i:372-4 (1962).

160. Galton, M. M., "The Epidemiology of Leptospirosis in the United States," Pub. Hlth. Rep., 74:141-148 (1959).

161. Garber, W. F., "Bacteriological Standards for Bathing Waters," Sewage and Industrial Wastes, 28:795-804 (1956).

162. Garber, W. F., "Critical Evaluation of Objectives and Standards of Bathing Water Bacteriological Quality." In Public Health Hazards of Microbial Pollution of Water, Proc. Rudolfs Research Conference, Rutgers, New Jersey, pp. 463-542 (1961).

163. Garrido-Morales, E., and O. Costa Mandry, "Typhoid Fever Spread by Water from a Cistern Contaminated by a Carrier," J. Prevent. Med., 5:351-358 (1931).

164. Geldreich, E. E., "Detection and Significance of Fecal Coliform Bacteria in Stream Pollution Studies," J. Water Poll. Cont. Fed., 37:1722-1726 (1965).

165. Geldreich, E. E., "Sanitary Significance of Fecal Coli Forms in the Environment," U. S. Dept. Interior, FWPCA Publication WP-20-3, 122 pp. (1966).

166. Geldreich, E. E., "Status of Bacteriological Procedures Used by State and Municipal Laboratories for Potable Water Examination," Health Lab. Sci., 4:9-16 (1967).

167. Geldreich, E. E., "Origins of Microbial Pollution in Streams," In *Transmission of Viruses by the Water Route*, G. Berg, Ed., John Wiley & Sons, New York (1967).

168. Geldreich, E. E., "Fecal Coliform Concepts in Stream Pollution," Water and Sewage Works, 114:R-98, 107-110 (1967).

169. Geldreich, E. E., and H. F. Clark, "Distilled Water Suitability for Microbiological Application," J. Milk and Food Technol., 28:351-358 (1965).

170. Geldreich, E. E., and N. A. Clarke, "Bacterial Pollution Indicators in the Intestinal Tract of Freshwater Fish," Appl. Microbiol., 14:429-437 (1966).

171. Geldreich, E. E., H. F. Clark, and C. B. Huff, "A Study of Pollution Indicators in a Waste Stabilization Pond," J. Water Poll. Cont. Fed., 36:1372-1379 (1964).

172. Geldreich, E. E., H. L. Jeter, and J. A. Winter, "Technical Considerations in Applying the Membrane Filter Procedure," Health Lab. Sci., 4:113-125 (1967).

173. Geldreich, E. E., P. W. Kabler, and H. F. Clark, "A Simplified Eosin Methylene Blue Agar," Pub. Health Lab., 14:78-83 (1956).

174. Geldreich, E. E., B. A. Kenner, and P. W. Kabler, "Occurrence of Coliforms, Fecal Coliforms and Streptococci on Vegetation and Insects," Appl. Microbiol., 12:63-69 (1964).

175. Geldreich, E. E., H. F. Clark, C. B. Huff, and L. C. Best, "A Fecal Coliform-Organism Medium for the Membrane Filter Technique," J. Amer. Water Works Assoc., 57:208-214 (1965)

176. Geldreich, E. E., P. W. Kabler, H. L. Jeter, and H. F. Clark, "A Delayed Incubation Membrane Filter Test for Coliform Bacteria in Water," Am. J. Public Health, 45:1462-1466 (1955).

177. Geldreich, E. E., L. C. Best, B. A. Kenner, and D. J. van Donsel, "The Bacteriological Aspects of Stormwater Pollution," J. Water Poll. Cont. Fed., 40:1861-1872 (1968).

178. Geldreich, E. E., R. A. Bordner, C. B. Huff, H. F. Clark, and P. W. Kabler, "Type Distribution of Coliform Bacteria in the Feces of Warm-Blooded Animals," J. Water Poll. Cont. Fed., 34:295-301 (1962).

179. Geldreich, E. E., H. F. Clark, P. W. Kabler, C. B. Huff, and R. H. Bordner, "The Coliform Group. II. Reactions in EC Medium at 45°C.," Appl. Microbiol., 6:347-355 (1958).

180. Geldreich, E. E., C. B. Huff, R. H. Bordner, P. W. Kabler, and H. F. Clark, "The Faecal Coli-Aerogenes Flora of Soils From Various Georaphical Areas," J. Appl. Bacteriol., 25:87-93 (1962).

181. Geldreich, E. E., "Applying Bacteriological Parameters to Recreational Water Quality," J. Amer. Water Works Assoc., 62:113-120 (1970).

182. Gelfand, H. M., "The Occurrence in Nature of the Coxsackie and ECHO Viruses," Progr. Med. Virol., 3:193-201 (1961).

183. Gevandan, P., and J. Tamalet, "Etude sur la Pollution des plages et Baignades sur le Littoral Mediterraneen," Riv. d'Hyg. Med. Sociale, 4:270-276 (1956).

184. Gilcreas, F. W., and S. M. Kelly, "Significance of the Coliform Test in Relation to Intestinal Virus Pollution of Water," J. New Eng. Water Works Assoc., 68:255-277 (1954).

185. Gilcreas, F. W., and S. M. Kelly, "Relation of Coliform-Organism Test to Enteric Virus Pollution," J. Am. Water Works Assoc., 47:683-694 (1955).

186. Gillespie, R. W., and J. Ryno, "Epidemiology of Leptospirosis," Amer. J. Pub. Hlth., 53:950-955 (1963).

187. Gilliam, A. G., "Epidemiology in Noncommunicable Disease," Pub. Hlth. Rep., 69:907-913 (1954).

188. Glantz, P. J. and G. E. Krantz, "Escherichia coli Serotypes Isolated from Fish and Their Environment," Health Lab. Sci., 2:54-63 (1965).

189. Goldberg, H. S., et al., "Leptospirosis in Random and Select Populations," Arch. Environ. Hlth., 10:21-23 (1965).

190. Gordon, J. E., "Medical Ecology and the Public Health," DSW-560 Amer. J. Med. Sci., 235:337=359 (1958).

191. Gordon, J. E., and T. H. Ingalls, "Progress of Medical Science--Preventive Medicine and Epidemicology: Acute Diarrheal Disease,' Amer. J. Med. Sci., 248:345-65 (1964).

192. Gordon, J. E., M. Pehar and N. S. Scrimshaw, "Acute Diarrheal Disease in Less Developed Countries," Bull. Wld. Hlth. Org., 31:29-32 (1964).

193. Gorman, A. E., and A. Wolman, "Waterborne Outbreaks in the U.S. and Canada and Their Significance," J. Amer. Water Works Assoc., 31:225-232 (1939).

194. Grabow, W.O.K., "The Virology of Wastewater Treatment," Water Research, 2:675-701 (1968).

195. Gray, J.D.A., "The Isolation of B. paratyphosus B from Sewage," Brit. Med. J., 1:142-149 (1929).

196. Green, C. E., and P. J. Beard, "Survival of E. typhi in Sewage Treatment Plant Processes," Am. J. Pub. Health, 28:762-781 (1938).

197. Green, D. M., "Waterborne Outbreak of Viral Gastroenteritis and Sonne Dysentery," J. Hyg. (Camb.), 66:383-389 (1968).

198. Greenberg, A. E. "Survival of Enteric Organisms in Sea Water," Pub. Health Rep., 71:77-89 (1956).

199. Greenberg, B., "Persistence of Bacteria in the Developmental Stages of the Housefly. I. Survival of Enteric Pathogens in the Normal and Aseptically Reared Host," Amer. J. Trop. Med. Hyg., 8:405-411, 412-622 (1959).

200. Greenberg, B., "Experimental Transmission of Salmonella typhimurium by Houseflies to Man," Amer. J. Hyg., 80:149-156 (1964).

201. Grinstein, S., J. L. Melnick, C. Wallis, "Virus Isolations from Sewage and from a Stream Receiving Effluents of Sewage Treatment Plants," Bull. Wld. Hlth. Org., 42:291-296 (1970).

202. Hagan, T. L., M. Pasternack, and G. C. Scholtz, "Waterborne Fluorides and Mortality," Pub. Hlth. Rep., 69:450-454 (1954).

203. Haldane, J. J., "The Influence of High Air Temperatures," J. Hyg. 5:494 (1905) cited by Hatch, T. F., in Industrial Hygiene and Toxicology, F. A. Patty, Ed. Interscience, N. Y. (1958)

204. Halliday, C. H., and M. D. Beck, "Typhoid Fever Epidemic, Santa Ana, California," J. Prevent. Med., 2:49-60 (1928).

205. Hanes, N. B., and R. Fragola, "Effect of Seawater Concentration on Survival of Indicator Bacteria," J. Water Poll. Cont. Fed., 39:97-104 (1967).

206. Hanes, N. B., G. A. Delaney, and C. J. O'Leary, "Relationship Between Escherichia coli, Type 1, Coliform and Enterococci in Water," J. Boston Soc. Civil Engr., 52:129-132 (1965).

207. Hanes, N. B., W. B. Sarles, and G. A. Roblich, "Dissolved Oxygen and Survival of Coliform Organisms and Enterococci," J. Am. Water Works Assoc., 56:441-450 (1964).

208. Hanks, T. G., "Literature Survey of Solid Waste/Disease Relationships," Final Report to the Office of Solid Wastes, U.S. Public Health Service, Aerojet-General Report No. 3337 (1967).

209. Hansen, H. G., and B. B. Berger, "Where Does Research Stand in Water Pollution Control," J. Water Poll. Cont. Fed., 33:477-484 (1961).

210. Hardy, A. V., "Diarrhoeal Diseases of Infants and Children--Mortality and Epidemiology," Bull. Wld. Hlth. Org., 21:309-319 (1959).

211. Harvey, R. W., and W. P. Phillips, "An Environmental Survey of Bakehouses and Abattoirs for Salmonellae," J. Hyg. (comb.), 59:93-103 (1961).

212. Hatch, T. F., "Heat Control in the Hot Industries," in Industrial Hygiene and Toxicology, Frank A. Patty, Editor, Interscience, N. Y. (1958).

213. Havens, W. P., Jr., !Properties of the Etiologic Agent of Infectious Hepatitis," Proc. Soc. Exp. Biol. Med., 58:203-212 (1945).

214. Hayes, W. J., W. F. Durham, and C. Cueto, Jr., "The Effect of Known Repeated Oral Doses of Chlorphenothane (DDT) in Man," J. Am. Med. Assoc., 162:890-897 (1956).

215 Hayes, W. J., Jr., G. E. Quinby, K. C. Walter, J. W. Elliott, and W. M. Upholt, "Storage of DDT and DDE in People with Different Degrees of Exposure to DDT," Am. Med. Assoc., Arch. Ind. Health, 18:398-406 (1958).

216. Healy, W. A., and R. P. Grossman, "Waterborne Epidemic at Keene, N. H.," J. New Eng. Water Works Assoc., 75:37-43 (1961).

217. Healy, W. A. and R. P. Grossman, "Typhoid is Still Waterborne," Water Works Engr., 114:38-41 (1961).

218. Heath, C. W., A. D. Alexander, and M. M. Galton, "Leptospirosis in the United States," New Eng. J. Med., 273:857-864 (1965).

219. Hedstrom, C. E., and E. Lycke, "An Experimental Study on Oysters as Virus Carriers," Amer. J. Hyg., 79:134-142 (1964).

220. Hendricks, C. W., "Bacterial Multiplication in Clear Mountain Water," Dissertation Abst., 27:4231 (1967); Water Poll. Abst. (Brit.), 41:196 (1968).

221. Hersey, D. F. and E. D. Shaw, "Viral Agents in Hepatitis," Lab. Invest., 19:558-572 (1968).

222. Heukelekian, H., and M. Albanese, "Enumeration and Survival of Human Tubercle Bacilli in Polluted Waters," Sew. Ind. Wastes, 28:9-19; 1094-1102 (1956).

223. Heukelelian, H., and H. B. Schulhoff, "Studies on Survival of *B. Typhosus* in Surface Waters and Sewage," Bulletin No. 589, New Jersey Agric. Exp. Station (1935).

224. Hind, H. W., and F. M. Goyan, "A New Concept of the Role of Hydrogen-ion Concentration and Buffer Systems in the Preparation of Ophthalmic Solutions," J. Am. Pharm. Assoc. (Sci. Ed.), 36:33-41 (1947).

225. Hind, H. W., and F. M. Goyan, "The Hydrogen-ion Concentration and Osmotic Properties of Lacrimal Fluid," J. Am. Pharm. Assoc. (Sci. Ed.), 38:477-479 (1949).

226. Hitchcock, G., D. A. J. Tyrell, and M. L. Bynoe, "Vaccination of Man with Attenuated Live Adenovirus," J. Hyg. Comb. 58:277-282 (1960).

227. Hoadley, A. W., "The Occurrence and Behavior of *Pseudomonas aeruginosa* in Surface Waters. Dissertation Abs., 27:4231; Water Poll. Abs., (Brit.), 41:551 (1968).

228. Hoadley, A. W., "On the Significance of *Pseudomonas aeruginosa* in Surface Waters," J. New Eng. Water Works Assoc., 82:99-111 (1968).

229. Hodges, R. E., "The Toxicity of Pesticides and Their Residues in Food," Nutrition Rev., 23:225-230 (1965).

230. Hoffman, W. S., W. I. Fishbein, and M. B. Andelman, "Pesticide Content of Human Fat Tissue," Arch. Environ. Health, 9:387-394 (1964).

231. Hoffman, W. S., H. Adler, W. I. Fishbein, and F. C. Bauer, "Relation of Pesticide Concentrations in Fat to Pathological Changes in Tissues," Arch. Environ. Health, 15:758-765 (1967).

232. Hong, et al, "Cardiac Rhythm During Diving," J. Appl. Physiol., 23:18-22 (1967).

233. Hornick, R. B., T. E. Woodward, F. R. McCrumb, M. J. Snyder, A. T. Dawkins, J. T. Bulkeley, F. De La Macorra, and F. A. Carozza, "Study of Induced Typhoid Fever in Man. I. Evaluation of Vaccine Effectiveness," Trans. Assoc. of Am. Physicians, 79:361-367 (1966).

234. Hornick, R. B., S. E. Greisman, T. E. Woodward, H. L. Dupont, A. T. Dawkins, and M. J. Snyder, "Typhoid Fever: Pathogenesis and Immunologic Control," New Eng. J. Med. 283, 13:686-691 (1970).

235. Horstmann, D., "Epidemiology of Poliomyelitis and Allied Diseases," Yale J. Biol. Med., 36:5-26 (1963).

236. Huebner, R. J., J. A. Bell, W. P. Rowe, T. G. Ward, R. G. Suskind, J. W. Hartley, and R. S. Paffenbarger, Jr., "Studies of Adenoidal-Pharyngeal-Conjunctival Vaccines in Volunteers," J. Amer. Med. Assoc., 159:986-989 (1955).

237. Hueper, W. C., "Carcinogens in the Human Environment," Arch. Path., 71:237-267, 355-380 (1961).

238. Hueper, W. C., and W. W. Payne, "Carcinogenic Effects of Adsorbates of Raw and Finished Water Supplies, "Amer. J. Clin. Path., 39:475-481 (1963).

239. Hutchison, D., R. H. Weaver, and M. Scherago, "The Incidence and Significance of Micro-organisms Antagonistic to E. coli in Water," J. Bact., 45:29-40 (1943); Water Pollution Abst., 16:(March, 1943).

240. Hueper, W. C., and W. W. Payne, "Observations on the Occurrence of Hepatomes in Rainbow Trout," J. Nat. Cancer Inst., 27:1123-1143 (1961).

241. Insalata, N. F., S. J. Schulte, and J. H. Berman, "Immunofluorescence Technique for Detection of Salmonellae in Various Foods," Appl. Microbiol., 15:1145-1149 (1967).

242. Jackson, G. G., H. F. Dowling, and W. J. Mogabgab, "Infectivity and Interrelationships of 2060 and JH Viruses in Volunteers," J. Lab & Clin. Med., 55:331-341.

243. Jellison, W. L., D. C. Epler, E. Kuhns, and G. M. Kohls, "Tularemia in Man from a Domestic Rural Water Supply," Pub. Health Rep., 65:1219-1225 (1950).

244. Jenkins, S. H. "Sewage, Trade Wastes and River Pollution," Soc. Chem. Ind., London, 46:40-51 (1961).

245. Jensen, K. A., and K. E. Jensen, "Occurrence of Tubercle Bacilli in Sewage, and Experiments on Sterilization of Tubercle Bacilli Containing Sewage with Chlorine," Acta Tuberc. Scand., 16:217-230 (1942).

246. Jeter, H. L., E. E. Geldreich, and H. F. Clark, "Type Differentiation of Coliform Organisms with Membrane Filter Technique," J. Am. Water Works Assoc., *46*:386-394 (1954).

247. Johnston, J. M., "Parathion Poisoning in Children," J. Pediat., *42*:286-291 (1953).

248. Johnston, M. A., and F. S. Thatcher, "Selective Inhibition of *Escherichia coli* in the presence of *Salmonella Typhimurium* by Phenethyl B-d-galactopyranoside," Appl. Microbiol., *15*:1223-1228 (1967).

249. Joyce, G., and H. H. Weiser, "Survival of Enteroviruses and Bacteriophage in Farm Pond Waters," J. Am. Water Works Assoc., *59*:491-501 (1967).

250. Kabler, P. W., "Bacterial Parameters of Water Quality. In *Water Quality Measurement and Instrumentation*," Proc. of the 1960 Seminar R. A. Taft San. Engr. Center, Cincinnati (1961).

251. Kabler, P. W., "Purification and Sanitary Control of Water (potable and waste)," Ann. Rev. Microbiol., *16*:127-138 (1962).

252. Kabler, P. W., "Bacteria can be a Nuisance," Chem. Eng. Prog., *59*:23-25 (1963).

253. Kabler, P. W., "Microbial Considerations in Drinking Water," J. Am. Water Works Assoc., *60*:1173-1180 (1968).

254. Kabler, P. W., H. F. Clark, and N. A. Clarke, "Pathogenic Microorganisms and Water-borne Disease," In *Proc. Rudolfs Research Conf. on Public Health Hazards of Microbial Pollution of Water*, Rutgers, New Brunswick, N. J., pp 9-56 (1961).

255. Kabler, P. W., H. F. Clark, and E. E. Geldreich, "Sanitary Significance of Coliform and Fecal Coliform Organisms in Surface Water," Pub. Hlth. Rep., *79*:58-65 (1964).

256. Kabler, P. W., and J. F. Kreissl, "Biological and Radiological Properties of Water," Farmstead Water Quality Improvement Seminar (Conf. Proc. 5-7 Oct. 1966) Am. Soc. Ag. Eng., St. Joseph, Michigan (1966).

257. Kabler, P. W., and H. F. Clark, "Coliform Group and Fecal Coliform Organisms as Indicators of Pollution in Drinking Water." J. Am. Water Works Assoc., *52*:1577-1582 (1960).

258. Kabler, P. W., S. L. Chang, N. A. Clarke, and H. F. Clark, "Pathogenic Bacteria and Viruses in Water Supplies," Univ. Ill. Bull. Circular 81, Bull., *61*:72-78 (1963).

259. Kapikian, A. Z., R. M. Chanock, T. E. Reichelderfer, T. G. Ward, R. J. Huebner, and J. A. Bell, "Inoculation of Human Volunteers with Parainfluenze Virus Type 3," J. Amer. Med. Assoc., 178:537-541 (1961).

260. Kapsenberg, J. G., "Salmonellae in Treated Sewage," Nederl. Tijdschr. v. Geneesk, 102:863 (1956).

261. Karalekas, P. C., and J. P. Lynch, "Recreational Activities of Springfield, Mass., Water Reservoirs, Past and Present," J. New Eng. Water Works Assoc., 79:18-21 (1965).

262. Kay, L. A., "The Construction and Operation of Open-Air Swimming Pools and Bathing Places," Canad. J. Pub. Health, 51:411-423 (1960).

263. Keatinge, W. R., "Death After Shipwreck," Brit. Med. J. 2:1537-1541 (1965).

264. Keatinge, W. R., "Survival in Cold Water: The Physiology and Treatment of Immersion Hypothermia and of Drowning," 135 pp. Oxford, Blackwell Scientific Publications, London (1969).

265. Kehr, R. W., and C. T. Butterfield, "Notes on the Relation Between Coliforms and Enteric Pathogens," Pub. Health Rep., 58:589-607 (1943).

266. Kehr, R. W., W. C. Purdy, J. B. Lackey, O. R. Placak, and W. E. Burns, "A Study of the Pollution and Natural Purification of the Scioto River," Pub. Health Bull. No. 276, Govt. Print. Off. (1941).

267. Kelly, C. B., and W. Arcisz, "Survival of Enteric Organisms in Shellfish," Pub. Hlth. Rep., 69:1205-1210 (1954).

268. Kelly, S. M. "Detection and Occurrence of Coxsackie Viruses in Sewage," Am. J. Pub. Hlth., 43:1532-1539 (1953).

269. Kelly, S. M., and W. W. Sanderson, "Chlorination of Polio Virus," Science, 126:560-563 (1957).

270. Kelly, S. M., and W. W. Sanderson, "The Effect of Chlorine in Water on Enteric Viruses," Am. J. Pub. Hlth., 48:1323-1334 (1958).

271. Kelly, S., and W. W. Sanderson, "The Effect of Sewage Treatment on Viruses," Sewage and Ind. Wastes, 31:683-699 (1959).

272. Kelly, S., and W. W. Sanderson, "Density of Enterovirus in Sewage," J. Water Poll. Cont. Fed., 32:1269-1273 (1960).

273. Kelly, S. M., and W. W. Sanderson, "The Effect of Chlorine in Water on Enteric Viruses. II. The Effect of Combined Chlorine in Poliomyelitis and Coxsackie Viruses," Am. J. Pub. Hlth., 50:14-20 (1966).

274. Kelly, S. M., and W. W. Sanderson, "Enteric Viruses in Wading Pools," Pub. Hlth. Rep., 76:199-207 (1961).

275. Kelly, S. and M. E. Clark, and M. B. Coleman, "Demonstration of Infectious Agents in Sewage," Amer. J. Pub. Hlth., 45:1438-46 (1955).

276. Kelly, S. M., W. W. Sanderson, and C. Neidl, "Removal of Enteroviruses from Sewage by Activatied Sludge," J. Water Poll. Cont. Fed., 33:1056-1063 (1961).

277. Kelly, S. J. Winser, and W. Winkelstein, Jr., "Poliomyelitis and Other Enteric Viruses in Sewage," Amer. J. Pub. Hlth., 47:72-77 (1957).

278. Kenner, B. A., H. F. Clark, and P. W. Kabler, "Fecal Streptococci. II. Quantification of Streptococci in Feces," Am. J. Pub. Hlth., 50:1553-1565 (1960).

279. Kenner, B. A., H. F. Clark, and P. W. Kabler, "Cultivation and Enumeration of Streptococci in Surface Waters," Appl. Microbiol., 9:15-20 (1961).

280. Khait, K. B., "Pollution of Littoral Waters and Sanitary Measures to Protect Them," Hyg. and Sanit., Moscow, 25:6-9 (1960); (cited by McKee, 1963).

281. Kiefer, D. M., "The Future Business," Chem. Eng. News, August, pp. 62-75 (1969).

282. Kissling, R. E., Laboratory Status of the Infectious Hepatitis Virus. In Transmission of Viruses by the Water Route (edited by G. Berg), John Wiley, New York (1967).

283. Kling, C., "In Search of Poliomyelitis Virus in Drinking Water," Intern. Bull. Econ. Med. Res. and Public Health, A40:161-175 (1939-40).

284. Knight, V., P. J. Gerone, W. R. Griffith, R. B. Couch, T. R. Cate, K. M. Johnson, D. J. Lang, H. E. Evans, A. Spickard, and J. A. Kasel, "Studies in Volunteers with Respiratory Viral Agents," Conf. on Newer Respiratory Disease Viruses. Amer. Rev. Resp. Dis. ** (Sept. Suppl.):135-143 (1963).

285. Knight, V., Moderator, Combined Clinical Staff Conference at the National Institutes of Health, "New Research on Influenza: Studies With Normal Volunteers." Annals of Internal Med. 62:1307-1325 (1965).

286. Koprowski, H., S. African Med. J., 29:1134 (1955); (cited by Plotkin and Katz, 1967).

287. Koprowski, H., Am. J. Trop. Med. Hyg., 5:440 (1956): (cited by Plotkin and Katz, 1967).

288. Korn, G. A., and T. M. Korn, Mathematical Handbook, McGraw Hill, N. Y., (1968).

289. Kraus, P., and G. Weber, "Investigations on the Viability of Pathogenic Organisms in Water Supplies and Surface Water," Zbl. Bakt., I., 171:509-523 (1958).

290. Kristensen, M., and K. Skadhange, "Disinfection of Drinking Water with Hydrochloric Acid," Acta. Pathol. Microbiol. Scand. (Den.), 40:216-226 (1957).

291. Krugman, S., R. Ward, and J. P. Giles, "The Natural History of Infectious Hepatitis," Am. J. Med., 32:717-728 (1962).

292. Lamb, G. A., T. D. Y. Chin, and L. E. Searce, "Isolation of Enteric Viruses from Sewage and River Water in a Metropolitan Area," Amer. J. Hyg., 80:320-327 (1964).

293. Langmuir, A. D., "The Surveillance of Communicable Diseases of National Importance," New Eng. J. Med., 268:182-192 (1963).

294. Lanbusch, E. J., "Rationale and Accomplishments of Chlorine Disinfection. In Public Health Hazards of Microbial Pollution of Water, Proc. Rudolfs Research Conference, Rutgers, New Jersey, pp. 543-610 (1961).

295. Lattanzi, W. E., and E. W. Mood, "A Comparison of Enterococci and Escherichia coli as Indexes of Water Pollution," Sew. Ind. Wastes, 23:1154-60; C. A. 46:2723 (1951).

296. Lee, D., J. M. Symons, and G. G. Robeck, "Watershed Human-Use Level and Water Quality," J. Amer. Water Works Assoc., 62:412-422 (1970).

297. Lehr, E. L., and C. C. Johnson, Jr., "Water Quality of Swimming Places. A Review," Pub. Health Rep., 69:742-748 (1954).

298. Leininger, H. V., and L. S. McClesky, "Bacterial Indicators of Pollution in Surface Waters," App. Microbiol., 1:119-130 (1953).

299. Lendon, N. C., and R. D. MacKenzie, "Tracing a Typhoid Carrier by Sewage Examination," Sanitarian, Lond., 59:306 (1951).

300. Lennox, M., R. Harvey, and S. Thomson, "An Outbreak of Food Poisoning due to Salmonella typhimurium, with Observations on Duration of Infection," J. Hyg. (Camb.) 52:311-320 (1954).

301. Litzky, W., W. L. Mallmann, and C. W. Fifield, "Comparison of the Most Probable Numbers of *Escherichia coli* and *Enterococci* in River Waters," Am. J. Pub. Hlth., 45:1049-1055 (1955).

302. Lobel, H. O., and R. F. Robinson, "Epidemiologic Aspects of an Outbreak of Infectious Hepatitis in Albany, N. Y.," Amer. J. Pub. Hlth., 55:1176-1182 (1965).

303. Loften, C. B., S. M. Morrison, and P. D. Leiby, "The Enterobacteriaceae of Some Colorado Small Mammals and Birds, and Their Possible Role in Gastroenteritis in Man and Domestic Animals," Zoonoses Research, 1:227-293 (1962).

304. London Metropolitan Water Board, 1931; 1957-58. Cited by Kabler, et al., 1961.

305. Lord, S. M., Jr., "Bacterial Indices of Bathing Water Quality," Thesis, Tufts University (1968).

306. Lopez, S. R., "A Waterborne Epidemic of Typhoid Fever," Puerto Rico Health Bulletin, 4:1-8 (1940).

307. Lund, E., C. E. Hedstrom, and N. Jantzen, "Occurrence of Enteric Viruses in Wastewater After Activated Sludge Treatment," J. Water Poll. Contr. Fed., 41:169-174 (1969).

308. Lynn, W. R., "Systems Analysis for Solid Waste Problems," Proc. Nat. Conf. on Solid Waste Disposal, Amer. Pub. Works Assoc. Special Report, 29:69-74 (1964).

309. Mack, W. N., J. R. Fry, B. J. Riegle, and W. L. Mallmann, "Enterovirus Removal by Activated Sludge Treatment," J. Water Poll. Cont. Fed., 34:1133-1139 (1962).

310. Mackenthun, K. M., and W. M. Ingram, *Biological Associated Problems in Freshwater Environments*, U. S. Dept. of the Interior, Fed. Water Poll. Cont. Adm., 287 pp. (1967).

311. Magath, T. B., "Water Transmission of Infections with Special Reference to Amebiasis," J. Am. Water Works Assoc., 27:63-77 (1935).

312. Malherbe, H. H., and M. Strickland-Cholmley, "Quantitative Studies on Viral Survival in Sewage Purification Processes. In *Transmission of Viruses by the Water Route*, (edited by G. Berg), John Wiley & Sons, New York (1967).

313. Mallmann, W. L., "Water Quality Yardsticks," J. Am. Water Works Assoc., 45:917-925 (1953).

314. Mallmann, W. L., "Critical Evaluation of Enterococci." In *Public Health Hazards of Microbial Pollution of Water*, Proc. Rudolfs Research Conf., Rutgers, New Jersey, pp. 181-199 (1961).

315. Mallmann, W. L., and W. Litsky, "Survival of Selected Enteric Organisms in Various Types of Soils," Am. J. Pub. Health, 41:38-43 (1951).

316. Mallory, A., E. Belden, and P. Brachman, "The Current Status of Typhoid Fever in the United States and a description of an Outbreak," J. Inf. Dis., 119:673-676 (1969).

317. Manigold, D. B., and J. A. Schultze, "Pesticides in Water: Pesticides in Selected Western Streams - A Progress Report," Pest. Monit. J., 3:124-135 (1969).

318. Mason, J. O., and W. R. McLean, "Infectious Hepatitis Traced to the Consumption of Raw Oysters, an Epidemiologic Study," Amer. J. Hyg., 75:90-111 (1962).

319. Maxcy, K. F., "Principles and Methods of Epidemiology." In Rivers and Horsfall, Viral and Rickettsial Infections of Man, Lippincott (1959).

320. Maynard, J. E., "Infectious Hepatitis at Fort Yukon, Alaska--Report of an Outbreak, 1960-61," Amer. J. Pub. Hlth., 53:31-39 (1963).

321. McCabe, L. J., and T. W. Haines, "Diarrheal Disease Control by Improved Human Excreta Disposal," Pub. Hlth. Rep., 72:921-928 (1957).

322. McCarthy, J. A., "Critical Evaluation of Coliform Organisms, In Public Health Hazards of Microbial Pollution of Water, Proc. Rudolfs Res. Conf., Rutgers, New Jersey, pp. 123-164 (1961).

323. McCoy, J. H., "River Pollution: Bacteriological Aspects and Their Significance," J. Roy. Sec. Hlth., 83:154-162 (1963).

324. McCoy, J. H., "Salmonella in Crude Sewage Effluent and Sewage-Polluted Waters," Adv. Water Poll. Res., 1:205-226 (1964).

325. McCoy, J. H., "Salmonella in Crude Sewage, Sewage Effluent and Sewage-Polluted Natural Waters," Advances in Water Pollution Research, B. A. Southgate, Ed., The MacMillan Co., N. Y., pp 206-226, see especially Table I, page 207, (1964).

326. McCoy, J. H., "The Isolation of Salmonella," J. Appl. Bact., 25:213-224 (1962).

327. McCullough, N. B., and C. W. Eisele, "Experimental Human Salmonellosis. I. Pathogenicity of Strains of Salmonalla meleagridis and Salmonella anatum obtained from Spray-dried Whole Egg." J. Inf. Dis., 88:278-289 (1951).

328. McCullough, N. B., and C. W. Eisele, "Experimental Human Salmonellosis. II. Immunity Studies Following Experimental Illness with *Salmonella Meleagridis* and *Salmonella anatum*., J. Immunol., 66:595-608 (1951).

329. McCullough, N. B., and C. W. Eisele, "Experimental Human Salmonellosis. III. Pathogenicity of Strains of *Salmonella newport*, *Salmonella derby*, and *Salmonella bareilly* Obtained from Spray-Dried Whole Egg," J. Inf. Dis., 89:209-213 (1951).

330. McCullough, N. B., and C. W. Eisele, "Experimental Human Salmonellosis. IV. Pathogenicity of Strains of *Salmonella pullorum* Obtained from Spray-Dried Whole Egg," J. Inf. Dis., 89:259-265 (1951).

331. McGaughey, P. H., "Study on Protection of Water Resources of Lake Tahoe Basin (1963); (cited by Mackenthun and Ingram, 1967).

332. McKee, J. E., *100 Problems in Environmental Health*, Jones Composition Co. and Kirby Lithograph Co., Washington, D. C., 185 pp. (1961).

333. McKee, J. E., "The Impact of Industrial Wastes on the Water Quality Equation," Paper presented at the Conf. on Practical Solutions to the Water Quality Equation, Santa Ana River Basin Water Poll. Cont. Bd., 17 October, 1963.

334. McKee, J. E., "Water Pollution Control," Trusts and Estates, 103:42-45 (1964).

335. McKee, J. E. and H. W. Wolf, *Water Quality Criteria*, 2nd ed., The Res. Agency of Calif., State Water Quality Cont. Bd., No. 3-A, Sacramento, 548 pp. (1963).

336. McKinney, R., H. E. Langley, and H. P. Tomlinson, "Survival of *Salmonella typhosa* During Anaerobic Digestion," Sew. Ind. Wastes, 30:1469 (1958).

337. McLean, D. M., "Contamination of Water by Viruses," J. Amer. Water Works Assoc., 56:585-591 (1964).

338. McLean, D. M., "Transmission of Viral Infections by Recreational Water." In *Transmission of Viruses by the Water Route*, John Wiley & Sons, New York, pp. 25-36 (1967).

339. McMicael, F. C., and J. E. McKee, "Report on Waste Water Reclamation at Whittier Narrows," W. M. Keck Lab. Env. Hlth. Eng., Calif. Inst. Tech., Pasadena, also State Water Quality Control Board, Sacramento, Calif. (1965).

340. Melnick, J. L., and R. P. Dow, "Poliomyelitis in Hidalgo County, Texas, 1948. Poliomyelitis and Coxsackie Virus from Flies," Amer. J. Hyg., *58*:288-309 (1953).

341. Melnick, J. L., and L. J. Penner, "The Survival of Poliomyelitis and Coxsackie Viruses Following Their Ingestion by Flies," J. Exp. Med., *96*:255-271 (1952).

342. Melnick, J. L., J. Emmons, J. H. Coffey, and H. Schoof, "Seasonal Distribution of Coxsackie Viruses in Urban Sewage and Flies," Amer. J. Hyg., *59*:164-184 (1954).

343. Merrell, J. C. and P. C. Ward, "Virus Control at the Santee, Calif., Project," J. Am. Water Works Assoc., *60*:145-153 (1968).

344. Merrell, J. C., W. F. Jopling, R. F. Bott, A. Katko, and H. E. Pinter, Santee Recreation Project: Final Report. U.S. Dept. of Interior, Fed. Water Poll. Cont. Adm., Pub. No. WP-20-7, 165 pp., (1967).

345. Messerschmidt, T., and R. Wedemeyer, "Der Nachweis von Typhus und Paratyphusbacterien in Stadtischem Abwasser," Zentralbl. Bakt. I. Abt. Orig., *156*:489 (1951).

346. Metcalf, T. G., and W. C. Stiles, "The Accumulation of Enteric Viruses by the Oyster, *Crassostris veiginica*." J. Inf. Dis., *115*:68-83, (1965).

347. Metcalf, T. G., and W. C. Stiles, "Survival of Enteric Viruses in Estuary Waters and Shellfish. In *Transmission of Viruses by the Water Route* (edited by G. Berg), John Wiley & Sons, New York. (1967).

348. Metzler, D. F., R. L. Culp, H. A. Stoltenberg, R. L. Woodward, G. Walton, S. L. Chang, N. A. Clarke, C. M. Palmer, and F. M. Middleton, "Emergency Use of Reclaimed Water for Potable Supply at Chanute, Kansas," J. Am. Water Works Assoc., *50*:1021-1057 (1958).

349. Meyer, T. R., R. Phillips, H. E. Lind, and L. M. Board, "A Milk-borne Typhoid Outbreak Traced to Dairy Water Supply," J. Milk Tech., *4*:123; J. Am. Water Works Assoc., *35*:240 (1943 Abst.) (1941).

350. Meynell, G. G., and J. Maw, "Evidence for a Two-Stage Model of Microbial Infection," J. Hyg., Camb. *66*:273-279 (1968).

351. Meynell, G. G., and E. W. Meynell, "The Growth of Microorganisms *in vivo* with Particular Reference to the Relation Between Dose and Latent Period," J. Hyg., Camb., *56*:323-346 (1958).

352. Meynell, G. G., and T. Williams, "Estimating the Date of Infection from Individual Response Times," J. Hyg., Camb., 65:131-134 (1967).

353. Minkus, A. J., "Recreational Use of Reservoirs," J. New Eng. Water Works Assoc., 79:32-40 (1965).

354. Mom, C. P., and C. O. Schaeffer, "Typhoid Bacteria in Sewage and in Sludge," Sewage Works J., 12:715-725 (1940).

355. Mood, E. W., "Development and Application of High-Free Residual Chlorination in the Treatment of Swimming Pool Water," Am. J. Pub. Health, 43:1258-64. C. A. 47:12712 (1953).

356. Mood, E. W., "The Role of Some Physico-Chemical Properties of Water as Causative Agents of Eye Irritation of Swimmers. In Water Quality Criteria, FWPCA Nat. Tech. Advisory Committee Report, (1968).

357. Moore, B., "A Survey of Beach Pollution at a Seaside Resort," J. Hyg., Camb., 52:71-86 (1954a).

358. Moore, B., "Sewage Contamination of Coastal Bathing Waters," Bull. Hyg., 29:689-704 (1954b).

359. Moore, B., "The Risk of Infection Through Bathing in Sewage-Polluted Water." Proc. 1st Int. Conf. on Waste Disposal in the Marine Environ., Univ. California, Berkeley, 22-25 July 1959 (1959).

360. Moore, B., "Sewage Contamination of Coastal Bathing Waters in England and Wales. A Bacteriological and Epidemiological Study," J. Hyg., Camb., 57:435-472 (1959).

361. Moore, B., "The Risk of Infection Through Bathing in Sewage-Polluted Waters. In Waste Disposal in the Marine Environment, Proc. 1st Int. Conf., ed. by E. A. Pearson, Pergamon Press, New York (1960).

362. Morris, W., and R. H. Weaver, "Streptococci as Indices of Pollution in Well Waters,"

363. Mosely, J. W., "Infectious Hepatitis in Clearfield County, Pa., A Probably Waterborne Epidemic," Am. J. Med., 26:555-562 (1959a).

364. Mosley, J. W., "Water-borne Infectious Hepatitis," Med. Prog., 26-:703-707; 748-753 (1959b).

365. Mosley, J. W., "Transmission of Viral Diseases by Drinking Water." In Transmission of Viruses by the Water Route, John Wiley & Sons, New York, pp. 5-24 (1967).

366. Mosely, W. H., J. F. Speers, and T. D. Y. Chin, "Epidemiologic Studies of a Large Urban Outbreak of Infectious Hepatitis," Aer. J. Pub. Hlth., 53: 1603-1617 (1963).

367. Murtaugh, J., and R. L. Bunch, "Sterols as a Measure of Fecal Pollution," J. Water Poll. Cont. Fed., 39:404-409 (1967).

368. Muss, D. L., "Are our Criteria for Bathing Water Pollution Valid?" Civil Eng., 33:37-39 (1963).

369. Nakamura, M., R. L. Stone, and J. E. Krubsack, "Survival of Shigella in Sea Water," Nature, 203:213-214 (1964).

370. National Technical Advisory Committee on Water Quality Criteria, Water Quality Criteria, Report of National Technical Advisory Committe to the Secretary of Interior (1969).

371. Neefe, J. R., and J. Stokes, Jr., "An Epidemic of Infectious Hepatitis Apparently due to a Waterborne Agent. Epidemiologic Observations and Transmission Experiments in Human Beings," J. Am. Med. Assoc., 128:1063-75 (1945).

372. Neefe, J. R., J. Stokes, Jr., J. B. Baty, and J. G. Reinhold, "Disinfection of Water Containing Causative Agents of Infectious (Epidemic) Hepatitis," J. Am. Med. Assoc., 128:1076-1080 (1945).

373. Neefe, J. R., J. Stokes, Jr., J. B. Baty, "Inactivation of the Virus of Infectious Hepatitis in Drinking Water," Am. J. Pub. Health, 37:365-372 (1947).

374. Norman, N. N., and P. W. Kabler, "Bacteriological Study of Irrigated Vegetables," Sew. and Ind. Wastes, 25:605-615 (1953).

375. Novick, A. "Growth of Bacteria," Ann. Rev. Microbiol., 9:97-110 (1955).

376. Nussbaum, I., and R. M. Garver, "Survival of Coliform Organisms in Pacific Ocean Coastal Waters," Sew. Ind. Wastes, 27:1383-1395 (1955).

377. Okun, D. A., et al., "A Review of the Literature of 1964 on Wastewater and Water Pollution Control: Industrial Wastes," J. Water Poll. Cont. Fed., 37:735-799 (1964).

378. Okuno, G. "Vaccination with Egg Passage Measles Virus by Inhalation," Am. J. Dis. of Children, 103:211-214 (1962).

379. Orlob, G. T., "Viability of Sewage Bacteria in Sea Water," Sew. Ind. Wastes, 28:1147-1155 (1956).

380. Ormsby, H. L., "An Interim Report on Ocular Disease due to APC Viruses in Ontario," Canad. J. Pub. Hlth., 46:500-515 (1955).

381. Ormsby, H. L. and W. S. Aitchison, "Pharyngo-Conjunctival Fever due to Swimming Pool Contact," Canad. Med. Assoc. J., *73*:864-872 (1955).

382. Outdoor Recreation Resources Review Committee, *Outdoor Recreation for America*, U.S. Govt. Printing Office, Washington, D.C., 246 pp. (1962).

383. Palazzolo, A. Z. R., E. M. Ansiaume, R. H. Leiguardia, and O. A. Peso, "Investigation of Bacteria of the Genus *Salmonella* in Canal Water; Its Relation to Intestinal Disorders Among Children," Rev. abr. Sanit. Nac., B. Aires, *33*:128 (1954).

384. Parker, M. T., "Human and Animal Sources of Gastrointestinal Infection. (a) The Spread of Some Bowel Infections from Human Sources," J. Roy. San. Inst., *74*:847-855

385. Paul, J. R., and H. T. Gardner, "Epidemiological Aspects of Hepatitis in U.S. Troops in Germany, 1946-50," Amer. J. Med., 8:565-580 (1950).

386. Paul, J. R., and J. D. Trask, "Poliomyelitis Virus in Sewage," Science, *90*:258-259 (1939).

387. Paul, J. R., and J. D. Trask, "The Virus of Poliomyelitis in Stools and Sewage," J. Am. Med. Assoc., *116*:493-497 (1941).

388. Pearson, E. A., "An Investigation of the Efficacy of Submarine Outfall Disposal of Sewage and Sludge," Publication No. 14, State Water Pollution Control Board, Sacramento, California (1956).

389. Petersen, N. J., and V. D. Hines, "The Relation of Summertime Gastrointestinal Illness to the Sanitary Quality of the Water Supplies in Six Rocky Mountain Communities," Am. J. Hyg., *71*:314-320 (1960).

390. Plotkin, S. A., and M. Katz, "Minimal Infective Doses of Viruses for Man by the Oral Route," *Transmission of Viruses by the Water Route* (edited by G. Berg), John Wiley & Sons, New York (1967).

391. Plotkin, S. A., D. Cornfeld and T. H. Ingalls, Am. J. Dis. Children, *110*:381 (1965); Cited by Plotkin and Katz, 1967.

392. Popp, L., "Vorfluter: ein Reichhaltiges salmonellen Reservoir. (Receiving Waters: Abundant Salmonella reservoirs), Zbl. Bakt., I. Abt. Orig., *66*:90-94 (1956).

393. Poynter, S. F. R., "The Problem of Viruses in Water," J. Soc. Water Treat. and Exam. (Eng.) *17*:187-204 (1968).

394. Pozkanzes, D., and W. Baldenkopf, "Waterborne Infectious Hepatitis Epidemic from a Chlorinated Municipal Supply," Pub. Health Repts., 76:745-755 (1961).

395. Presnell, N.W., W. Arcisz, and C. B. Kelly, "Comparison of the MF and MPN Techniques in Examining Sea Water," Pub. Hlth. Rep., 69:300-304 (1954).

396. Press, E., "An Interstate Drowning Study," Am. J. Pub. Health, 58:2275-89 (1968).

397. Prier, J. E., and R. Riley, "Significance of Water in Natural Animal Virus Transmission." In *Transmission of Viruses by the Water Route* (edited by G. Berg), John Wiley & Sons, New York (1967).

398. Quinby, G., and A. B. Lemmon, "Parathion Residues as a Cause of Poisoning in Crop Workers," J. Am. Med. Assoc., 166:740-746 (1958).

399. Quinby, G. E., W. J. Hayes, Jr., J. F. Armstrong, and W. F. Durham, "DDT Storage in the United States Population," J. Am. Med. Assoc., 191:175-179 (1965).

400. Ravenholt, R. T., and S. P. Lehman, "History, Epidemiology and Control of Typhoid Fever in Seattle," Medical Times, 92:342-353 (1964).

401. Rawson, D. S., "The Physical Limnology of Great Slave Lake," J. Fish. Res. Bd., Canada, 8:1-66 (1950).

402. Reichelderfer, T. E., R. M. Chanock, J. E. Craighead, R. J. Huebner, H. C. Turner, W. James, T. G. Ward, "Infection of Human Volunteers with Type 2 Hemadsorption Virus," Science, 128:779-780 (1958).

403. Rent, A. I., "The Viability of Some Bacteria of the Intestinal Group in Water Containing Humus," Zdravoochr. Beloruss., 1:54-61; Water Poll. Abst., 31:144-150 (1958, abst.).

404. Rhodes, A. J., E. M. Clarke, D. S. Knowles, A. M. Goodfellow, and W. L. Donahue, "Prolonged Survival of Human Poliomyelitis Virus in Experimentally Infected River Water," Can. J. Pub. Hlth., 41:146-157, (1950).

405. Richardson, N. J., and V. Bokkenheuser, "Salmonellae and Shigellae in a Group of Periurban South African Bantu School Children," J. Hyg. (Camb.), 61:257-263 (1963).

406. Richter, Jr., "Salmonellen im Vorfluter; ein Ortliches Abwasserproblem," Stadthyg., 7:101 (1956).

407. Riegelmann, S., and D. G. Vaughan, Jr., "A Rational Basis for the Preparation of Ophthalmic Solutions. Part I. J. Am. Pharm. Assoc., (Pract. Pharm. Ed.), 19:474-477 (1958).

408. Riegelmann, S., D. G. Vaughan, Jr., and M. Okumoto, "Compounding Ophthalmic Solutions," J. Am. Pharm. Assoc. (Prac. Pharm. Ed.), 16:742-746 (1955).

409. Riley, C., "Plankton Studies. IV. Georges Bank," Bull. Bingham Oceanogr. Coll., 7:1-73 (1941).

410. Ritter, C., I. F. Shull, and R. L. Quinley, "Comparison of Coliform Group Organisms with Enterococci from Well Waters," Am. J. Pub. Health, 46:612-620 (1956).

411. Rittenberg, S. C., "Studies on Coliform Bacteria Discharged from the Hyperion Outfall," Final Bacteriological Report, 130 pp. Allan Hancock Foundation for Sci. Res., Univ. So. Calif., Los Angeles (1956).

412. Rivers, T. M. and F. L. Horsfall, "Viral and Rickettsial Infections in Man," Lippincott (1959).

413. Robeck, G. G., N. A. Clarke, and K. A. Dostal, "Effectiveness of Water Treatment Processes in Virus Removal," J. Amer. Water Works Assoc., 54:1275-1290 W62-49 (1962).

414. Robinson, C. R., F. W. Doane, and A. Rhodes, "Report of an Outbreak of Febrile Illness with Pharyngeal Lesions and Exanthem: Toronto, Summer 1957. Isolation of a Group A Coxsackie Virus," Can. Med. Assoc. J., 79:615-622 (1958).

415. Robinson, J., "The Estimation of Carrier Rate from Amebic Surveys," J. Hyg., Camb., 66:531-539 (1968).

416. Robinson, R. A., and K. I. Loken, "Age Susceptibility and Excretion of Salmonella typhimurium in Calves," J. Hyg., Camb., 66:207-216 (1968).

417. Rosen, G. "Human Health, Community Life, and Rediscovery of the Environment," Amer. J. Pub. Hlth., 54:1-6 (1964).

418. Roseberry, D. A., "Relationship of Recreational Use to Bacterial Densities of Forrest Lake," J. Am. Water Works Assoc., 56:43-59 (1964).

419. Rosenstein, B. J., "Shigella and Salmonella Enteritis in Infants and Children," Bull, Johns Hopkins Hospital, 115:407-415 (1964).

420. Ruchhoft, L. C., "Studies on the Longevity of Bacillus typhosus (Eberthella typhi) in Sewage Sludge," Sewage Works J., 6:1054-1062 (1934).

421. Rudolfs, W., L. L. Falk, and R. A. Ragatzkie, "Literature Review on the Occurrence and Survival of Enteric, Pathogenic, and Related Organisms in Soil, Water, Sewage, and on Vegetation, I. Bacterial and Virol Diseases," Sewage and Industrial Wastes, 22:1261-1275 (1950).

422. Ruskin, J., and J. S. Remington, "Immunity and Intracellular Infection: Resistance to Bacteria in Mice Infected with a Protozoan," Science, 160:72-73 (1968).

423. Russell, F. E., and P. Kotin, "Squamous papilloma in the White Croaker," J. Natl. Cancer Inst., 18:857-861 (1957).

424. Sabin, A. B., "Behavior of Chimpanzee-Avirulent Poliomyelitis Viruses in Experimentally Infected Human Volunteers," Am. J. Med. Sci., 230:1-8 (1955).

425. Sabin, A. B., "Cause and Control of Fatal Infantile Diarrheal Disease," Am. J. Trop. Med. Hyg., 12:556-66 (1963).

426. Sanderson, W. W., and S. Kelly, "Discussion of Paper, "Pathogenic Microorganisms and Waterborne Disease," In *Public Health Hazards of Microbial Pollution of Water*, Proc. Rudolfs Res. Conf., June 1961, Rutgers, New Brunswick, N. J., pp. 57-72 (1961).

427. Saphra, I., and J. W. Winter, "Clinical Manifestations of Salmonellosis in Man," New Eng. J. Med., 256:1128-34 (1957).

428. Savage, W., "Paratyphoid Fever: An Epidemiological Study," J. Hyg., Camb., 42:393-405 (1942).

429. Scott, W. J., "Classification of Inland and Shore Waters," Sew. Works J., 14:1064-1072 (1942).

430. Scott, W. J., "Sanitary Study of Shore Bathing Waters," Conn. Health Bull., 65:1-50 (1951).

431. Scott, W. J., "Recent Sanitary Study of Shore Bathing Waters," Conn. Health Bull., 67:139-148 (1953).

432. Scott, W. J., "A Two-Year Study of Shore Bathing Waters," Conn. Health Bull., 71:111-130 (1957).

433. Sherman, J. N., C. F. Niven, and K. L. Smiley, "*Streptococcus salivarius* and Other Non-Hemolytic Streptococci of the Human Throat," J. Bact., 45-249 (1943).

434. Sherwood, H. P., "Some Observations on the Viability of Sewage Bacteria in Relation to Self Purification of Mussels," Proc. Soc. Appl. Bacteriol., 15:21-35 (1952); Water Poll. Abst., 27:1528 (1954, Abst.).

435. Shimkin, M., B. K. Koe, and L. Zechmeister, "An Instance of the Occurrence of Carcinogenic Substances in Certain Barnacles," Science, 113:650-651 (1951).

436. Shrewsbury, J. F. D., and G. J. Barson, "A Note on the Absolute Viability in Water of S. typhi and the Dysentery Bacilli," Brit. Med. J., (1952):954; (abst.) J. Am. Water Works Assoc., 46:72 P & R (1954).

437. Shrewsbury, J. F. D., and G. J. Barson, "On the Absolute Viability of Certain Pathogenic Bacteria in a Synthetic Well-Water," Jour. Path. and Bact., 74:215-223 (1957).

438. Shulman, N. R. "Hepatitis-Associated Antigen," Amer. J. Med. 49:669-692 (1970).

439. Shuval, H. I., S. Cymbalista, B. Fatal, and N. Goldblum, "Concentration of Enteric Viruses in Water by Hydro-Extraction and Two-Phase Separation," In Transmission of Viruses by the Water Route, G. Berg, ed., John Wiley and Sons, New York, pp. 45-55 (1967).

440. Slanetz, L. W., 'The Detection and Use of Enterococci as Indicators of Water Pollution. In Public Health Hazards of Microbial Pollution of Water, Proc. Rudolfs Research Conf., Rutgers, New Jersey, pp. 200-234 (1961).

441. Slanetz, L. W., and C. H. Bartley, "Numbers of Enterococci in Water, Sewage and Feces Determined by the Membrane Filter Technique with an Improved Medium," J. Bacteriol., 74:591-595 (1957).

442. Slanetz, L. W., and C. H. Bartley, "Survival of Fecal Streptococci in Sea Water," Health Lab. Sci., 2:142-149 (1965).

443. Smith, H. W., "The Isolation of Salmonellae from the Mesenteric Lymph Nodes and Faeces of Pigs, Cattle, Sheep, Dogs, and Cats, and from Other Organs of Poultry," J. Hyg. (Camb.), 57:266-273 (1959).

444. Smith, H. W., "The Effect of Feeding Pigs on Food Naturally Contaminated with Salmonellae," J. Hyg. (Camb.), 58:381-389 (1960).

445. Smith, R. S., and T. D. Woolsey, "Bathing Water Quality and Health. II. Inland River." U.S. Public Health Service, Environmental Health Center, Cincinnati, Ohio (1952).

446. Smith, R. S., T. D. Woolsey, and A. H. Stevenson, "Bathing Water Quality and Health. I. Great Lakes," U.S. Public Health Service, Environmental Health Center, Cincinnati, Ohio (1951)

447. Smith, R. S., C. E. Sponagle, E. E. Geldreich, L. J. McCabe, and T. D. Woolsey, "Bathing Water Quality and Health. III. Coastal Water," U.S. Public Health Service, R. A. Taft Sanitary Engineering Center, Cincinnati, Ohio (1950).

448. Spino, D. F., "Elevated Temperature Technique for the Isolation of Salmonella from Streams," Appl. Microbiol., 14:591-596 (1966).

449. Stein, W. J., and W. J. Hayes, Jr., "Health Survey of Pest Control Operators," Indust. Med. Surg., 33:549-555 (1964)

450. Steiniger, F., "Salmonella panama in Harbor Water Polluted by Sewage," Arztl. Wschr., 2:42-60, (1952); Water Poll. Abst., 27:2314 (1954).

451. Steiniger, F., "Fish as Carriers of Bacteria," Desinfekt. u. Gesundtheitswes. 46:36-48, (1954); Water Poll. Abst., 28:2348 (1955).

452. Stevenson, A. H., "Water Quality Requirements for Recreational Uses," Sewage Works J., 21:110-119 (1949).

453. Stevenson, A. H., "Studies of Bathing Water Quality and Health," Am. J. Pub. Hlth., 43:529-538 (1953).

454. Stewart, A. D., and S. C. Ghosal, "On the Value of Wilson and Blair's Bismuth Sulfite Medium in the Isolation of Bact. typhosum from River Water," Ind. J. Med. Res., 25:591 (1938).

455. Stokinger, H. and R. L. Woodward, "Toxicologic Methods for Establishing Drinking Water Standards," J. Amer. Water Works Assoc., 50:515-529 (1958).

456. Streeter, H. W., and E. B. Phelps, "A Study of the Pollution and Natural Purification of the Ohio River," U.S. Public Health Service Bulletin Nos. 143, 146 (1925).

457. Streeter, H. W., "A Formulation of Bacterial Changes Occurring in Polluted Water," Sewage Works J., 6:208-215 (1934).

458. Streeter, H. W., "Tendencies in Standards of River and Lake Cleanliness," Sew. Works J., 6:721-727 (1934).

459. Streeter, H. W., "Limits of Pollution Loading for Water Purification Systems," J. Am. Water Works Assoc., 27:1-15 (1935).

460. Streeter, H. W., "Limiting Standards of Bacterial Quality for Sources of Purified Water Supplies," J. Am. Water Works Assoc., 27:1110-1120 (1935).

461. Streeter, H. W., "Standards of Stream Sanitation," Water and Sewage, 84:61-68 (1946).

462. Streeter, H. W., "Bacterial-Quality Objectives for the Ohio River. A Guide for the evaluation of sanitary condition of waters used for potable supplies and recreational uses," Ohio River Valley Water Sanitation Commission, Cincinnati, 26 pp. (1951).

463. Strobel, G. A., "Coliform-Fecal Coliform Bacteria in Tidal Waters," J. San. Eng. Div., Proc. Am. Soc. Civil Eng., 94:SA4)641-656 (1968).

464. Taylor, F. B., J. H. Eagen, H. F. D. Smith, and R. F. Coene, "The Case for Water-Borne Infectious Hepatitis," Am. J. Pub. Health, 56:2093-2100 (1966); Water Poll. Abst. (Brit.) 41:150 (1968).

465. Taylor-Robinson, D., "Para-Influenza and Virus Infections in Adult Volunteers," J. Hyg. Camb., 61:407-417 (1963).

466. Thompson, S., "The Number of Bacilli Harboured by Enteric Carriers," J. Hyg., Camb., 52:67-70 (1954).

467. Thompson, S., "The Number of Pathogenic Bacilli in Intestinal Diseases," J. Hyg., Camb., 53:217-224 (1955).

468. Tomkin, R. B., H. H. Weiser, and G. W. Malaney, "Salmonella and Shigella in Untreated Pond Water," J. Am. Water Works Assoc., 55:592-596 (1963).

469. Tonney, F. O., and R. E. Noble, "Colon-Aerogenes Types of Bacteria as Criteria of Fecal Pollution," J. Am. Water Works Assoc., 24:1267-1272 (1932).

470. Tonney, F. O., and R. E. Noble, "The Relation of Direct Bacterial Coli and Bacterial Aerogenes Counts to Sources of Pollution," J. Am. Water Works Assoc., 22:488-495 (1930).

471. Toomey, J. A., W. S. Takas, and H. M. Weaver, "Isolation of Poliomyelitis Virus from Creek Water by Direct Transmission to the Cotton Rat," Am. J. Dis. Child., 70:293-302 (1945).

472. U.S. Dept. Health, Education, & Welfare, Bureau of State Services, National Water Quality Network Annual Compilation of Data Oct. 1, 1957-Sept. 30, 1958.

473. U.S. Public Health Service, "Diseases of Animal Origin or Common to Man and Animals in the United States," USPHS, U.S. Dept. HEW, Communicable Disease Center, Atlanta, Georgia, 11 pp. (1963).

474. Vaccaro, R. F., M. P. Briggs, C. L. Carey, and B. H. Ketchum, "Viability of Escherichia Coli in Sea Water," Amer. J. Pub. Health, 40:1257-1265 (1950).

475. van der Holden, J., and E. Szenberg, "Leptospira Infections in Rats in Israel," Trop. Geoz. Med., 16:377-384 (1965).

476. van der Schaaf, A., and J. C. Atteveld, "Frequency of Salmonellae in Effluents of Modern Plants for Sewage Treatment and Possibilities for Improvement," J. Microbiol. and Serology (Neth.), 31:221 (1965) (ex. McKee, 1963).

477. van Oye, E., ed., *The World Problem of Salmonellosis*, Monographic Biological (den Haag) (1964).

478. Van Donsel, D. J., E. E. Geldreich, and N. A. Clarke, "Seasonal Variations in Survival of Indicator Bacteria in Soil and Their Contribution to Storm-Water Pollution," Appl. Microbiol., 15:1362-1370 (1967).

479. Vaughan, R. D. and G. L. Harlow, "Report on Pollution of the Detroit River, Michigan, Waters of Lake Erie, and Their Tributaries," U.S. Dept. Health, Educ. and Welfare, Div. Water Supply and Poll. Cont., Washington, D.C. (1965).

480. Veldee, M. V., "An Epidemiological Study of Suspected Waterborne Gastroenteritis," Am. J. Pub.Health, 21:1227-1235 (1931).

481. Verduin, J., "Primary Production in Lakes," Limnology and Oceanography, 1:85-91 (1956).

482. Verhoestraete, L. J., and R. R. Puffer, "Diarrheal Disease with Special Reference to the Americas," Bull. Wld. Hlth. Org., 19:23-51, (1958).

483. Viswanathan, R., et al., "Infectious Hepatitis in Delhi (1955-56): A Critical Study," Ind. J. Med. Res., 45:January, 1957 Supplementary number.

484. Walker, K. D., M. B. Goette, and G. S. Batchelor, "Dichlorodiphenyltrichlorethane and Dichlorodiphenyldichlorethylene Content of Prepared Meals," J. Agric. Food Chem., 2:1034-1037 (1954).

485. Wallace, C., and J. L. Melnick, "Detection of Viruses in Large Volumes of Natural Waters by Concentration on Insoluble Polyelectrolytes," Water Res. 4:787-796 (1970).

486. Wallace, E. C., "Infectious Hepatitis: Report of an Outbreak, Apparently Waterborne," Med. J. Australia, 45:101-102 (1958).

487. Walton, G., "Survey of Literature Relating to Infant Methemoglobinemia due to Nitrate-Contaminated Water," Amer. J. Pub. Hlth., 41:986-96 (1951).

488. Wang, W. L. L., S. G. Dunlop, and P. S. Munson, "Factors Inflencing the Survival of *Shigella* in Wastewater and Irrigation Water," J. Water Poll. Cont. Fed., 38:1775-1781 (1966).

489. Weber, G., "Salmonella in Water," Zentr. Bakteriol., 163:312 (1957). Reported in J. Am. Water Works Assoc., 50:68 (P&R) June 1958, taken from Water Pollution Abstracts.

490. Wedman, E. E., "Epidemiology--A Prerequisite to the Eradication of Animal Disease," J. Amer. Vet. Med. Assoc., 147: 1550-5 (1965).

491. Weibel, S. R., R. J. Anderson, and R. L. Woodward, "Urban Land Runoff as a Factor in Stream Pollution," J. Water Poll. Cont. Fed., 36:914-924 (1964) (W64-23).

492. Weibel, S. R., F. R. Dixon, R. B. Weidner, and L. J. McCabe, "Water-Borne Disease Outbreaks, 1946-60," J. Amer. Water Works Assoc., 56:947-58 (1964).

493. Weibel, S. R., R. B. Weidner, A. G. Christianson, and R. J. Anderson, "Characterization, Treatment, and Disposal of Urban Stormwater," 3rd Int. Conf. on Water Poll. Res., Munich, pp. 1-15 (1966).

494. Werner, S. B., P. H. Jones, W. M. McCormack, E. A. Ager, and P. T. Holme, "Gastroenteritis Following Ingestion of Sewage-Polluted Water," Am. J. Epidemiol., 82:277-285 (1969).

495. Weston, A. D., and G. P. Edwards, "Pollution of Boston Harbor," Proc. Amer. Soc. Civil Engrs., 65:383-390 (1939).

496. Whatley, T. R., G. W. Comstock, H. J. Garber, and F. S. Sanchez, Jr., "A Waterborne Outbreak of Infectious Hepatitis in a Small Maryland Town," Am. J. Epidem., 87:138-151 (1968).

497. Wiesner Report, "Use of Pesticides," U.S. Govt. Printing Office (LC63-61540) PR 35.8:ScI 2/P43, May 15, 1963 (1963).

498. Wiley, J. S., "Pathogen Survival in Composting Municipal Wastes," J. Water Poll. Cont. Fed., 34:80-90 (1962).

499. Wiley, J. S., T. D. Y. Chin, C. M. Gravelle, "Enterovirus in Sewage During a Poliomyelitis Epidemic," J. Water Poll. Cont. Fed., 34:168-178 (1962).

500. Wilson, W. J., 1928; 1933; 1938, "Studies on Enteric Bacteria in Sewage and Water," cited by Kabler, et al., 1961.

501. Wolff, J. W., "The Relation of Animal Hosts of Parasitic Leptospires in the Netherlands with Human Leptospirosis," Trop. Geog. Med., 1:2-8 (1965).

502. Wolfe, H. R., W. F. Durham, K. C. Walker, and J. F. Armstrong, "Health Hazards of Discarded Pesticide Containers," Arch. Environ. Hlth., 3:45-51 (1961).

503. Woodward, R. L., "Significance of Pesticides in Water Supplies," J. Am Water Works Assoc., 52:1367-1372 (1960).

504. Woodward, R. L., "Review of the Relation of Water Quality and Simple Goiter," J. Amer. Water Works Assoc., 55:887-896 (1963).

505. Wang, W., and D. J. Brabec, "Nature of Turbidity in the Illinois River," J. Amer. Water Works Assoc. (Sept), 460-464 (1969).

506. Yeary, R. A., "Public Health Significance of Chemical Residues in Foods," J. Am. Vet. Assoc., 149:145-150 (1966).

507. Zapp, J. A., Jr., "The Toxicologic Evaluation of Low-Level Long Term Exposure of Pesticides in Man," In Research in Pesticides, Academic Press, Inc., New York, pp. 305-313 (1965).

508. ZoBell, C. E., "The Occurrence of Coliform Bacteria in Ocenic Water," J. Bact., 42:284-292 (1941).

B. BIBLIOGRAPHY OF PESTICIDES AND OILS

BIBLIOGRAPHY

1. Adlung, K. G., "The Toxicity of Insecticidal and Acaricidal Agents to Fish," Naturwissenschaften, **44**:471 (1957).

2. Akhmedov, B. K., and V. B. Danilov, "Morphological, Pathological, and Histochemical Changes in the Organs of Rats During Long-Term Exposure to Metaphos and Carbophos (Malathion) Vapors," Med. Zhur. Uzbek, 17:8; C. A. 71:69633s.

3. Aldridge, W. N., Biochem. J. 53:110, in O'Brien, *Insecticides*, p. 71 (1953).

4. Aldridge, W. N., Biochem. J. 53:117; in O'Brien, *Insecticides*, p. 71 (1953).

5. Andrews, G. C., and A. N. Domonkos, *Diseases of the Skin*, W. B. Saunders Co. 69-75 (1960).

6. Anon., "Toxic Effects of Benzene Hexachloride and its Principal Isomers," Amer. Med. Assoc. **147**:571 (1951); in McKee, *Water Quality Criteria*, p. 360.

7. Anon., "Outlines of Information on Pesticides, Part I, Agricultural Fungicides," J. Amer. Med. Assoc. **157**:237 (1955); in *Water Quality Criteria*, p. 389.

8. Anon., "Sacramento River Water Pollution Survey," Appendix B. Water Quality Bul. No. 111, Calif. Dept. of Water Resources, p. 87 (1962).

9. Anon., "Pace Quickens for DDT Restrictions," Chem. Eng. News, 48(4):32-3 (1970).

10. Anon., "HEW, USDA Hold Firm; 2, 4-D Ruling Postponed," Chem. Eng. News 48(7):11-12 (1970).

11. Anon., *Report of the Committee on Water Quality Criteria*, Federal Water Pollution Control Administration, 234 pp (1968).

12. Anon., "Methoxychlor Carcinogenic," Food-Drug-Cosmetic Law Jour., **13**:4-6 (1958).

13. Anon., "Vietnam Defoliant Seen Causing Birth Defects," London Sunday Times, quoted by Los Angeles Times, Nov. 30 (1969).

14. Anon., "Bacterial-Quality Objectives for the Ohio River," Ohio River Valley Water Sanitation Commission (1951).

15. Anon., "Monitoring Agricultural Pesticide Residues, 1965-1967. A final Report on Soil, Crops, Water, Sediment, and Wildlife in Six Study Areas," Agricultural Research Service. U.S.D.A., ARS 81-32, (1969).

16. Anon., "Pesticides and Public Health," Pesticides Program, National Communicable Disease Center, Publ. Health Service, U. S. Dept. Health, Educ. Welf. 16 pp. (1967).

17. Anon., "Water Quality Criteria," Report of the National Technical Advisory Committee to the Secretary of the Interior, 234 pp. (1968).

18. Anon., "Parathion Poisoning," J. Amer. Med. Assoc., 171:2126 (1959).

19. Anon., "Pesticide Levels Low in West Coast Fish," Chem. & Eng. News, 42:71 (1964).

20. Anon., "Pesticide Residue Problems Probed," Chem. & Eng. News, 42:32-33 (1964).

21. Antommaria, P., "Airborne Particulates in Pittsburgh: Association with p, p'-DDT," Science, 150:1476-77 (1965).

22. Antonyuk, O. K., "Some Data From a Primary Toxicological Study of the Herbicides Dachal and Zectran," Gig. Toksikol. Pestits. Klin. Otravlenii (4):244-5 (1966); C. A. 69:42940m.

23. Ariens, A. Th., E. M. Cohen, E. Meeter, and O. L. Wolthuis, "Reversible Necrosis in Striated Muscle Fibers of the Rat After Severe Intoxication with Various Cholinesterase Inhibitors," Ind. Med. Surg., 37:845-7 (1968). C. A. 70:85817q.

24. Arterberry, J. D., R. W. Binifaci, E. W. Nash, and G. E. Quinby, "Potentiation of Phosphorus Insecticides by Phenothiazine Derivatives," J. Amer. Med. Assoc., 185:110-112. 185:110-12 (1962).

25. Bailey, T. E., and J. R. Hannum, "Distribution of Pesticides in California," J. San. Engr. Division, 93:27-43 (1967).

26. Ball, W. L., J. W. Sinclair, M. Crevier, and K. Kay, "Modification of Parathion's Toxicity for Rats by Pretreatment with Chlorinated Hydrocarbon Insecticides," Can. J. Biochem. Physiol., 32:440-445 (1954).

27. Bann, J. M., T. J. Decino, N. W. Earle, and Y. P. Sun, J. Agr. Food Chem., 4:937; in O'Brien, Insecticides, p. 140 (1958).

28. Baroni, C., G. J. von Esch, and U. Saffioti, "Carcinogenesis Tests of Two Inorganic Arsenicals," Arch. Environ. Health, 7:668-73 (1963).

29. Baylor, C. H., and N. K. Weaver, "A Health Survey of Petroleum Asphalt Workers," Arch. Environ. Health, *17*:210-14 (1968).

30. Beard, R. L., J. Econ. Entom., *42*:292; in O'Brien, *Insecticides*, p. 149 (1949).

31. Bengelsdorf, I., "DDT Turns Body's Guardians Into Indiscriminate Killers," Los Angeles Times, Dec. 4, (1969).

32. Berlin, M., L. G. Jerksell, and H. von Ubisch, "Uptake and Retention of Mercury in the Mouse Brain," Arch. Environ. Health, *12*:33-42 (1966).

33. Biden-Steele, K., and R. E. Stuckey, Lancet p. 235 (August 17, 1946).

34. Bingham, E., and H. L. Falk, "Environmental Carcinogens. The Modifying Effect of Cocarcinogens on the Threshold Response," Arch. Environ. Health, *19*:779-83 (1969).

35. Bingham, E., A. W. Horton, and R. Tye, "The Carcinogenic Potency of Certain Oils," Arch. Environ. Health, *19*:449-51 (1965).

36. Bland, W. F., and R. L. Davidson, *Petroleum Processing Handbook*, McGraw-Hill, New York (1967).

37. Bloom, S. C., and S. E. Degler, "Pesticides and Pollution," Environmental Management Series, Bureau of National Affairs (1969).

38. Bogusz, M., "Activity of Certain Enzyme Systems in Agricultural Workers Exposed to Organophosphorus Insecticides," Pol. Tyg. Lek. *23*:786-9; C. A. 70:27974c (1968).

39. Boyd, E. M., and C. J. Krijnen, "Toxicity of Captan and Protein Deficient Diet," J. Clin. Pharmacol. J. New Drugs, *8*:225-34; C. A. 69:26259r (1968).

40. Breed, A. W., and J. J. Lichty, "DDT and Dieldrin in Rivers; A Report of the National Quality Network," Science, *14*:899-90 (1963).

41. Breidenbach, A. W., "Pesticide Residues in Air and Water," Arch. Environ. Health, *10*:827-30 (1965).

42. Breidenbach, A. W., and J. J. Lichtenberg, "DDT and Dieldrin in Rivers: A Report of the National Water Quality Network," Science, *141*:899-901 (1963).

43. Brooks, G. T., Nature, *186*:96; in O'Brien, *Insecticides*, p. 141 (1960).

44. Brooks, G. T., and A. Harrison, Biochem. Pharmacol., 13:827; in O'Brien, Insecticides, p. 144 (1964).

45. Brooks, G. T., A. Harrison, and G. T. Cox, Nature, 197:311; in O'Brien, Insecticides, p. 143-4 (1963).

46. Brown, A. W. A., Insect Control By Chemicals, Wiley, New York (1951).

47. Bulla, C. D., III, and E. Edgerley, Jr., "Photochemical Degradation of Refractory Organic Compounds," Jour. Water Pollution Control Federation, 40:546-56 (1968).

48. Campbell, J. E., L. A. Richardson, and M. L. Schafter, "Insecticide Residues in the Human Diet," Arch. Environ. Health, 10:831-6 (1965).

49. Candura, F., "Pathogenesis of Poisoning by Industrial Solvents (With Special Regard to Benzene)," Ann. Med. (Milan), 12:112-6; C. A. 69:79953u (1968).

50. Case, R. A. M., "Toxic Effects of 2,2-bis(p-Chlorphenyl) 1,1,1,-Trichlorethane (DDT) in Man," Brit. Med. J., 842-845 (Dec. 15, 1945).

51. Casida, J. E., and K. B. Augustinsson, Biochem. Biophys. Acta., 36:411; in O'Brien, Insecticides, p. 97 (1959).

52. Casida, J. E., K. B. Augustinsson, and G. Johnson, J. Econ. Entomol., 53:205; in O'Brien, Insecticides, p. 97 (1960).

53. Ceretoo, F., and A. M. Cosseddu, "Prolonged Administration of Parathion to Lactating Sheep. I. Cholinesterase Activity, Hematocrit Value, and Histopathological Picture of Treated Animals," Vet. Ital., 20:3-31; C. A. 71:59154x (1969).

54. Chang, S. C., and C. W. Kearns, J. Econ. Entom., 57:397; in O'Brien, Insecticides, p. 171 (1964).

55. Chernov, O. V., and I. I. Khitsenko, "Blastomogenic Properties of Some Derivatives of Dithiocarbamic Acid (Herbicides, Zineb and Ziram), Vop. Oncol., 15:71-4; C. A. 71:21105u (1969).

56. Chin, Y., and C. Tant, "The Effect of DDT on Cutaneous Sensations in Man," Science, 103:654 (1946).

57. Cleveland, F. P., "Summary of Work on Aldrin and Dieldrin Toxicity at the Kettering Laboratory," Arch. Environ. Health, 13:195-8 (1966).

58. Cohen, J. M., L. J. Kamphake, A. E. Lemke, C. Henderson, and R. L. Woodward, "Effect of Fish Poisons on Water Supplies. Part I. Removal of Toxic Materials," Jour. Amer. Water Wks. Ass. 52:1551 (1960).

59. Cohen, J. M., G. M. Rourke, and R. L. Woodward, "Effect of Fish Poisons on Water Supples. II. Odor Problems," Jour. Amer. Water Wks. Ass., 53:49-62 (1961).

60. Cohen, J. M., and C. Pinkerton, "Widespread Translocation of Pesticides by Air Transport and Rainout," Adv. in Chemistry Series No. 60, ACS (1966).

61. Cohen, A. J., and J. N. Smith, Nature, 189:600; in O'Brien, Insecticides, p. 140-1 (1961).

62. Cueto, C., and J.H.U. Brown, "Chemical Fractionationation of Adrenocorticolytic Drug," Endocrinology, 62:326-333 (1958).

63. Cueto, C., J.H.U. Brown, and A. P. Richardson, Jr., "Biological Studies of Adrenocorticolytic Agent and Isolation of Active Components," Endocrinology, 62:334-9 (1958).

64. Curley, A., M. F. Copeland, and R. D. Kimbrough, "Chlorinated Hydrocarbon Insecticides in Organs of Stillborn and Blood of Newborn Babies," Arch. Environ. Health, 19:628 (1969).

65. Curley, A., and L. K. Garretson, "Acute Chlordane Poisoning. Clinical and Chemical Studies," Arch. Environ. Health, 18:211-15 (1969).

66. Czerkinski, S. N., Protection of Surface Waters From Pollution by Industrial Waste Waters. Lit. Ber. Wass. Abwass. Luft. Boden, 4:98; in McKee, Water Quality Criteria p. 368 (1955).

67. Dale, W. E., M. F. Copland, and W. J. Hayes, Jr., "Chlorinated Insecticides in the Body Fat of People in India," Bull. World Health Organ., 33:471 (1965).

68. Dale, W. E., T. B. Gaines, and W. J. Hayes, Jr., and G. W. Pearce, "Poisoning by DDT: Relation Between Clinical Signs and Concentration in Rat Brain," Science, 142:1474-6 (1963).

69. Dale, W. E., and G. E. Quinby, "Chlorinated Insecticides in the Body Fat of People in the United States," Science, 142:593-5 (1963).

70. Dangerfield, W. G., "Toxicity of DDT to Man," Brit. Med. J. p. 27 (Jan. 5, 1946).

71. Davies, J. E., W. F. Edmundson, N. J. Schneider, and J. C. Cassady, "Pesticides in People," Pest. Monit. J., 2:80-85 (1968).

72. Davis, J. E., J. H. Davis, D. E. Frazier, J. B. Mann, and J. C. Welke, "Urinary p-Nitrophenol Concentrations in Acute and Chronic Parathion Exposures," Organic Pesticides in the Environment, Advances in Chemistry Series, American Chemical Society, 60:67-78 (1966).

73. Davis, K. J., and O. G. Fitzhugh, "Tumorigenic Potential of Aldrin and Dieldrin for Mice," Toxicol. Appl. Pharmacol., 4:187-9 (1962).

74. Dean, E. L., "Progress Report on Water Quality Criteria," Jour. Amer. Water Wks. Ass., 54:1313-35 (1962).

75. Denes, A., "Problems of Food Chemistry Concerning Residues of Chlorinated Hydrocarbons," Naehrung, 6:48 (1962).

76. Dinman, B. D., "Acute Combined Toxicity Due to DDVP and Chlordane," Arch. Environ. Health, 9:765-69 (1964).

77. DuBois, K. P., "Insecticides, Rodenticides, Herbicides, Household Hazards," Postgrad. Med., 24:278 (1958).

78. DuBois, K. P., "Low-Level Organophosphate Residues in the Diet," Arch. Environ. Health, 10:837-41 (1965).

79. Duggan, R. E., H. C. Barry, and L. Y. Johnson, "Pesticide Residues in Total-Diet Samples," Science, 151:101-4 (1966).

80. Duggan, R. E., and G. Q. Lipscomb, "Dietary Intake of Pesticide Chemicals in the United States (II) June 1966-April 1968," Pest. Monit. J., 2:153-162 (1969).

81. Dunstan, A. E., A. W. Nash, B. T. Brooks, and H. Tizard, eds. The Science of Petroleum, Oxford Univ. Press, London, v. 2, 935-1057 (1938).

82. Durham, W. F., "Pesticide Exposure Levels in Man and Animals," Arch. Environ. Health, 10:842-6 (1965).

83. Durham, W. F., "Physiological Effects of Pesticide Use," Jour. Amer. Water Wks. Ass., 57:1311-18 (1965).

84. Durham, W. F., J. F. Armstrong, and G. E. Quinby, "DDT and DDE Content of Complete Prepared Meals," Arch. Environ. Health, 11:641-7 (1965).

85. Durham, W. F., "Pesticide Residues in Foods in Relation to Human Health," Residue Reviews, 4:34-81 (1963).

86. Durham, W. F., J. F. Armstrong, and G. E. Quinby, "DDA Excretion Levels," Arch. Environ. Health, 11:76-79 (1965).

87. Durham, W. F., J. F. Armstrong, W. M. Upholt, and C. Heller, "Insecticide Content of Diet and Body Fat of Alaskan Natives," Science, 134:1880 (1961).

88. Dutkiewicz, T., and H. Tyras, "Skin Absorption of Toluene, Xylene, and Styrene by Man," Brit. Jour. Ind. Med., 25:243; C.A. 69:79980a (1968).

89. Earle, N. W., J. Agr. Food Chem., 11:281; in O'Brien, Insecticides, p. 141 (1963).

90. Edmundson, W. F., and J. E. Davies, "Occupational Dermatitis From Naled," Arch. Environ. Health, 15:89-91 (1967).

91. Edson, E. F., "The Effects of Prolonged Administration of Small Daily Doses of Parathion in the Rat, Pig and Man," Report - Fisons Pest Control Ltd. (1957); Cited in Hays Annal N.Y. Acad. Sc., 160 (1969).

92. Edson, E. F., et al., "No-Effect Levels of Three Organophosphates in the Rat, Pig and Man," Food Cosmet. Toxicol., 2:311-316 (1964).

93. Ellis, E., Chemistry of Petroleum Derivatives," Reinhold Pub. Co., New York (1934).

94. Emerson, T. E., G. M. Brake, and L. B. Hinshaw, Can. J. Physiol. Pharmacol., 42:41; in O'Brien, Insecticides, p. 137

95. Epstein, E., and W. J. Grant, "Chlorinated Insecticides in Run-Off Water as Affected by Crop Rotation," Soil Sci. Soc. Amer. Proc., 32:423-6; C.A. 69:43013y (1968).

96. Falk, H. L., S. J. Thompson, and P. Kotin, "Carcinogenic Potential of Pesticides," Arch. Environ. Health, 10:847-58 (1965).

97. Faust, S. D., "Pollution of the Water Environment by Organic Pesticides," Clinical Pharmacoloty and Therapeutics, 5:677 (1964).

98. Faust, S. D., and O. M. Aly, "Water Pollution by Organic Pesticides," Jour. Amer. Water Wks. Ass., 56:267-79 (1964).

99. Fisher, A., Contact Dermatitis, Lea and Febiger, Philadelphia (1967).

100. Fitzhugh, O. G., and A. A. Nelson, "Chronic Oral Toxicity of DDT," J. Pharmacol. Exp. Therap., 89:18-30 (1947).

101. Frawley, J. P., R. Weir, T. Tusing, K. P. DuBois, and J. C. Calandra, "Toxicologic Investigations on Delnav," Toxic. Appl. Pharmacol., 5:605-624 (1963).

102. Fukushima, M., "Histopathological Effects of Naphthalene on Rabbit Spleen," Showa Igakkai Zasshi, 28:144-51; C.A. 69:75292e (1968).

103. Funckes, A. J., G. R. Hayes, Jr., and W. V. Hartwell, "Urinary Excretion of Paranitrophenol by Volunteers Following Dermal Exposure to Parathion at Different Ambient Temperatures," Ag. and Food Chem., 11:455-457 (1963).

104. Gage, S. D., "The Control of Oil Pollution in Rhode Island," Jour. Boston Soc. Civil Eng., 11:237(1924); in J. E. McKee, Water Quality Criteria, p. 232 (1963).

105. Ganelin, R. S., C. Cueto, and G. A. Mail, "Exposure to Parathion," Jour. Am. Med. Ass., 188:807-10 (1964).

106. Ganelin, R. S., G. A. Mail, and C. Cueto, Jr., "Hazards of Equipment Contaminated With Parathion," Arch. Environ. Health, 8:826-28 (1964).

107. Gatrilevskaya, L. N., V. P. Laskina, and E. V. Faidysh, "Experimental Basis for the Permissible Concentration of Sevin in Reservoir Waters," Prom. Zagryazneniya Vodoemov (8):33-45; C.A. 69:34978q (1967).

108. Gerarde, H. W., "Toxicological Studies on Hydrocarbons. IX. The Aspiration Hazard and Toxicity of Hydrocarbons and Hydrocarbon Mixtures," Arch. Environ. Health, 6:329-41 (1963).

109. Gianotti, O., R. L. Metcalf, and R. B. March, Ann. Entomol. Soc. Am., 49:588; in O'Brien, Insecticides, p. 139 (1956).

110. Glauser, S. C., E. M. Glauser, and B. F. Rusy, "Influence of Gas Density and Viscosity on the Work of Breathing," Arch. Environ. Health, 19:654-60 (1969).

111. Gleason, M. N., R. E. Gosselin, H. C. Hodge, and R. P. Smith, Clinical Toxicology of Commercial Products. Williams and Wilkins, Baltimore (1969).

112. Goldblatt, M. W., "Organic Phosphorus Insecticides and the Antidotal Action of Atropine," Pharm. J., 164:229-233 (1950).

113. Gol'dshtein, N. I., "Normalization of Saifos (Menazon) in Reservoirs," Gig. Toksikol. Pestits. Klin. Otravleni No. 4:298-9; C.A. 69:51163n (1966).

114. Goldwater, L. J., M. B. Jacobs, and A. C. Ladd "Absorption and Excretion of Mercury in Man. I. Relationship of Mercury in Blood and Urine," Arch. Environ. Health, 4:537-41 (1962).

115. Goldwater, L. J., M. B. Jacobs, and A. G. Ladd, "Absorption and Excretion of Mercury in Man. IV. Tolerance to Mercury," Arch. Environ. Health, 7:568-73 (1963).

116. Goldwater, L. J. and M. M. Joselow, "Absorption and Excretion of Mercury in Man. XIII. Effects of Mercury Exposure on Urinary Excretion of coproporphyrin and Delta-Aminolevulimic Acid," Arch. Environ. Health, 15:327-31 (1967).

117. Goldwater, L. J., A. C. Ladd, and M. B. Jocobs, "Absorption and Excretion of Mercury in Man. VII. Significance of Mercury in Blood," Arch. Environ. Health, 9:735-41 (1964).

118. Goldwater, L. J., and A. Nicolau, "Absorption and Excretion of Mercury in Man. IX. Persistence of Mercury in Blood and Urine Following Cessation of Exposure," Arch. Environ. Health, 12:196-8 (1966).

119. Gontea, I., N. Nicolau, and E. Bistriceanu, "Influence of Chronic Toluene Poisoning on Vitamin C in the Rat," Igiena, 17(2)65-8; C.A. 69:75281a (1968).

120. Good, E. E., and G. W. Ware, "Effects of Insecticides on Reproduction in the Laboratory Mouse. IV. Endrin and Dieldrin," Toxicol. Appl. Pharmacol., 14:201-3; C.A. 70:86554p (1969).

121. Gowdey, C. W., A. R. Graham, J. J. Seguin, and G. W. Stavraky, Can. J. Biochem. Physiol., 32:498; in O'Brien, Insecticides, p. 136-7 (1954).

122. Gowdey, C. W., A. R. Graham, J. J. Seguin, G. W. Stavraky, and R. A. Waud, Can. J. Med. Sci., 30:533; in O'Brien, Insecticides, p. 136-7 (1952).

123. Gowdey, C. W., and G. W. Stavraky, Can. J. Biochem. Physiol. 33:272; in O'Brien, Insecticides, p. 136-7 (1955).

124. Green, R. S., C. G. Gunnerson, and J. J. Lichtenberg, "Pesticides in Our National Waters," Publ. Amer. Ass. Advan. Sci. 85:137-56; C.A. 70:31501p (1967).

125. Grob, D., and A. M. Harvey, "Observations of the Effect of Tetraethyl Pyrophosphate (TEPP) in Man and on its Use in the Treatment of Myasthenia Gravis," Bull. Hopkins Hosp., 84:532-567 (1949).

126. Gunther, F. A., W. E. Westlake, and P. S. Jaglan, "Reported Solubilities of 738 Pesticide Chemicals in Water," Residue Rev. 20:(1968).

127. Halpern, L. K., W. E. Wooldridge, and R. S. Weiss, "Appraisal of the Toxicity of the Gamma Isomer of Hexachlorocyclohexane in Clinical Usage," Arch. Derm. and Syph., 62:648-650 (1950).

128. Hanlon, J. J., "Letters to the Editor," Arch. Environ. Health, 12:676 (1966).

129. Hapke, H. J., "Toxicology of the Herbicide Simazine," Berlin Muenchen Tierarztl. Wochenschr., 81:301-3; C.A. 69:85621a (1968).

130. Harris, P. L., "Prolonged Ingestion of Liquid Petrolatum," J. Am. Med. Ass., 187:1041 (1964).

131. Hartwell, W. V., G. R. Hayes, Jr., and A. J. Funckes, "Respiratory Exposure to Volunteers of Parathion," Arch. Environ. Health, 8:820-25 (1964).

132. Hathaway, D. E., "The Biochemistry of Dieldrin and Telodrin," Arch. Environ. Health, 11:380-8 (1965).

133. Hayes, G. R., A. J. Funckes, and W. V. Hartwell, "Dermal Exposure of Human Volunteers to Parathion," Arch. Environ. Health, 8:829-33 (1964).

134. Hayes, W. J., Jr., "Occurrence of Poisoning by Pesticides," Arch. Environ. Health, 9:621-5 (1964).

135. Hayes, W. J., Jr., "Review of the Metabolism of Chlorinated Hydrocarbon Insecticides Especially in Mammals," Ann. Rev. Pharmacol., 5:27 (1965).

136. Hayes, W. J., Jr., "Monitoring Food and People for Pesticide Content," Scientific Aspects of Pest Control, Publ. No. 1402 National Academy of Sciences-National Research Council, 314-342 (1966).

137. Hayes, W. J., Jr., "Sweden Bans DDT," Arch. Environ. Health, 18:872 (1969).

138. Hayes, W. J., Jr., W. E. Dale, and R. L. LeBreton, "Storage of Insecticides in French People," Nature, 199:1189-91 (1963).

139. Haves. W. J., Jr., W. F. Durham, and C. Cueto, "Effects of Known Repeated Oral Doses of Chlorophenothane (DDT) in Man," J. Amer. Med. Assoc., 162:890-7 (1956).

140. Hayes, W. J., Jr., and C. I. Pirkle, "Mortality From Pesticides in 1961," Arch. Environ. Health, 12:42-55 (1966).

141. Hayes, W. J., Jr., G. E. Quinby, K. C. Walker, J. W. Elliot, and W. M. Upholt, "Storage of DDT and DDE in People With Different Degrees of Exposure to DDT," A.M.A. Arch. Ind. Health, 18:398-406 (1958).

142. Hayes, W. J., Jr., "Pesticides and Human Toxicity," Annal. N.Y. Acad. Sci., 160:40-51 (1969).

143. Hayes, W. J., Jr., "Toxicity of Pesticides to Man: Risks From Present Levels," Proc. Roy. Soc. London, 167B:101-127 (1967).

144. Hill, K. R., and G. Robinson. "A Total Case of DDT Poisoning in a Child," Brit. Med. J. 845-846 (Dec. 15, 1945).

145. Hodges, R. E., "The Toxicity of Pesticides and Their Residues in Food," Nutrition Reviews, 23:225 (1965).

146. Hoffman, W. S., W. I. Fishbein, and M. B. Andelman, "The Pesticide Content of Human Fat Tissue," Arch. Environ. Health, 9:387-94 (1964).

147. Hoffman, W. S., W. I. Fishbein, and M. B. Andelman, "Pesticide Storage in Human Fat Tissue," J. Am. Med. Assoc., 188: 819 (1964).

148. Hoffman, W. S., H. Adler, W. I. Fishbein, and F. C. Bauer, "Relation of Pesticide Concentration in Fat to Pathological Changes in Tissues," Arch. Environ. Health, 15:758-765 (1967).

149. Holden, A. V., "A Study of the Absorption of ^{14}C-Labeled DDT From Water by Fish," Ann. Appl. Biol., 50:467-477 (1962).

150. Holmberg, R. E., and V. H. Ferm, "Interrelationships of Selenium, Cadmium, and Arsenic in Mammalian Teratogenesis," Arch. Environ. Health, 18:873 (1969).

151. Hoogendam, I, J.P.J. Versteeg, and M. de Vlieger, "Nine Years' Toxicity Control in Insecticide Plants," Arch. Environ. Health, 10:441-48 (1965).

152. Hoover, A. W., and L. J. Goldwater, "Absorption and Excretion of Mercury in Man. X. Dental Amalgams as a Source of Urinary Mercury," Arch. Environ. Health, 12:506-8 (1966).

153. Hruban, Z., S. Schulman, N. E. Warner, K. P. DuBois, S. Bunnag, and S. C. Bunnag, "Hypoglycemia Resulting From Insecticide Poisoning," J. Am. Med. Assoc., 184:590-2 (1963).

154. Hsieh, H. C., "DDT Intoxication in a Family of Southern Taiwan," Indust. Hyg. Occup. Med., 10:344-346 (1954).

155. Hucker, H. B., J. R. Gillette, and B. B. Brodie, J. Pharmacol. Expt. Therap., 129:94; in O'Brien, Insecticides, p. 155 (1960).

156. Hunter, C. G., "Allowable Human Body Concentrations of Organochlorine Pesticides," Med. Lav., 59:577-83; C.A. 71: 21158p (1968).

157. Hunter, C. G., and J. Robertson, "Pharmacodynamics of Dieldrin (HEOD). I. Ingestion by Human Subjects for 18 Months," Arch. Environ. Health, 15:14-26 (1967).

158. Hunter, G. G., J. Robinson, and K. W. Jager, "Aldrin and Dieldrin. The Safety of Present Exposures of the General Populations of the United Kingdom and the United States," Food Cosmetol. Toxicol., 5:781-7; C.A. 69:9907h (1967).

159. Hunter, C. G., J. Robinson, and M. Roberts, "Pharmacodynamics of Dieldrin (HEOD). II. Ingestion by Human Subjects for 18-24 Months and Post-Exposure for Eight Months," Arch. Environ. Health, 18:12-21 (1969).

160. Hutterer, F., F. M. Klion, A. Wengraf, F. Schaffner, and H. Popper, "Hepatocellular Adaptation and Injury. Structural and Biochemical Changes Following Dieldrin and Methyl Butter Yellow," Lab. Invest., 20:455-64; C.A. 70:114135y (1969).

161. Iuchi, I., Bull. Yamaguchi Med. School, 6:1; in O'Brien, Insecticides, p. 66 (1958).

162. Ivanova-Chemishanka, L., G. Dimova, N. Mosheva-Izmirova, M. Sheitanov, "Changes in Thyroid Gland After a Month of Intoxication with Zineb," Khig. Zdraveopazvane, 11:555-9; C.A. 71:48654j (1968).

163. Jaakmes, V., "Experimental Data for the Hygienic Basis of Permissible Concentrations of Gasoline Produced From Shale Oil," Tr. Tsent. Inst. Usovershe. Vrachei, 103:23-7; C.A. 69:69521n (1967).

164. Jacobs, M. B., and L. J. Goldwater, "Absorption and Excretion of Mercury in Man. VIII. Mercury Exposure From House Paint - A Controlled Study on Humans," Arch. Environ. Health, 11:582-7 (1965).

165. Jacobs, M. B., A. C. Ladd, and L. J. Goldwater, "Absorption and Excretion of Mercury in Man. III. Blood Mercury in Relation to Duration of Exposure," Arch. Environ. Health, 6:634-7 (1963).

166. Jacobs, M. B., A. C. Ladd, and L. J. Goldwater, "Absorption and Excretion of Mercury in Man. VI. Significance of Mercury in Urine," Arch. Environ. Health, 9:455-63 (1964).

167. Johnston, J. M., "Parathion Poisoning in Children," J. Pediat., 42:286-291 (1953).

168. Jones, A. T., and E. O. Longley, "Mercury Exposure in a Jewelry Molding Process," Arch. Environ. Health, 13:769-75 (1966).

169. Joselow, M. M., and L. J. Goldwater, "Absorption and Excretion of Mercury in Man. XII. Relation Between Urinary Mercury and Proteinuria," Arch. Environ. Health, 15:155-8 (1967).

170. Joselow, M. M., L. J. Goldwater, A. Alvarez, and J. Herndon, "Absorption and Excretion of Mercury in Man. XV. Occupational Exposure Among Dentists," Arch. Environ. Health, 17: 39-43 (1968).

171. Joselow, M. M., R. Ruiz, and L. J. Goldwater, "Absorption and Excretion of Mercury in Man. XIV. Salivary Excretion of Mercury and its Relation to Blood and Urine Mercury," Arch. Environ. Health, 17:35-7 (1968).

172. Judah, J. D., Brit. J. Pharmacol., 4:120; in O'Brien, Insecticides, p. 125 (1949).

173. Kanagaratnam, K., W. H. Boon, and T. K. Hoh, "Parathion Poisoning From Contaminated Barley," Lancet 1:538-542 (1960).

174. Kailin, E. W., "Pesticide Storage: How Much Change?" J. Am. Med. Assoc., 191:188 (1965).

175. Kashin, L. M., I. L. Kulinskaya, and L. F. Mikhailovskaya, "Changes in Animals Under the Influence of Small Concentrations of Xylene," Vrach. Delo (8):109-12; C.A. 69:85107n (1968).

176. Katz, M., et al., "Biology: A Review of the Literature of 1964. Waste Water and Water Pollution Control," J. Water Poll. Contr. Fed., 37:899-905 (1965).

177. Kay, J. H., R. J. Palozzolo, and J. C. Calondra, "Subacute Dermal Toxicity of 2,4-D." Arch. Environ. Health, 11:648-51 (1965).

178. Kay, K., "Effect of Pesticides on Enzyme Systems in Mammals. Organic Pesticides in the Environment," Advances in Chemistry Series, American Chem. Society, 60:54-66 (1966).

179. Keane, W. T., Jr., and M. R. Zavon, "Validity of a Critical Blood Level for Prevention of Dieldrin Intoxication," Arch. Environ. Health, 19:36-44; C.A. 71:59115k (1969).

180. Khokhol'kova, G. A., and A. G. Pestova, "Accumulation and Distribution of Diptal (Trichloroallyl Diisopropylthiocarbamate) in Rats Following Ingestion," Vrach. Delo 99-102; C.A. 71: 59941b (1969).

181. Kleinman, G. D., I. West, and M. S. Augustine, "Health Hazards of Pesticides," J. Amer. Med. Assoc., 181:332-33 (1962).

182. Klimmer, O. R., "Application of Organotin Fungicides in Agriculture With Regard to Toxicity," Pflanzenschutzberichte, 37(4-6):57-66; C.A. 69:18227p (1968).

183. Koch, R. B., "Chlorinated Hydrocarbon Insecticides: Inhibition of Rabbit Brain ATPase Activities," J. Neurochem., 16:269-71; C.A. 70:76733g (1969).

184. Kojima, K., and R. D. O'Brien, Unpublished Observations; in O'Brien, Insecticides, p. 71, (1966).

185. Kossick, P., "Paraffin Poisoning," S. Africa Med. J., 35:112; in J. Amer. Med. Assoc., 176:462 (1961).

186. Krueger, H. R., and R. D. O'Brien, J. Econ. Entom., 52:1063; in O'Brien, Insecticides, p. 74-5 (1959).

187. Kunkle, S. H., and J. R. Meiman, "Water Quality of Mountain Watersheds," Colorado State University Hydrology Paper No. 21:53 (1967).

188. Kurbat, N. M., "Effect of Ethylenebisdithiocarbamates on the Blood and Hematopoietic Organs of Animals," Zdravookhr. Beloruss., 2:49-51; C.A. 71:48683t (1968).

189. Kutakov, K. V., "Comparative Sanitational-Toxicological Features of Thiophosphoric Acid Esters in Connection with Water Reservoir Sanitation," Gig. Sanit., 33(12):13-18; C.A. 70:70915w (1968).

190. Ladd, A. C., L. J. Goldwater, and M. B. Jacobs, "Absorption and Excretion of Mercury in Man. V. Toxicity of Phenylmercurials," Arch. Environ. Health, 9:43-52 (1964).

191. Laguna, W. D., "Chemical Quality of Water, Brookhaven National Laboratory and Vicinity New York," U. S. Geological Survey Bulletin 1156-D, Washington, U.S. Govt. Printing Office, 73 (1964).

192. Lai, P., and F. Virdis, "Prolonged Administration of Parathion to Lactating Sheep. II. Lambs and Dogs Fed Milk and Meat of Treated Sheep," Vet. Ital., 20(1-2):32-47; C.A. 71:59155y (1969).

193. Laug, E. P., F. M. Kunze, and C. S. Prickett, "Occurrence of DDT In Human Fat and Milk," A.M.A. Arch. Ind. Hygiene, 3:245-6 (1951).

194. Laug, E. P., A. A. Nelson, O. G. Fitzhugh, and F. M. Kunze, "Liver Cell Alteration and DDT Storage in the Fat of the Rat Induced by Dietary Levels of 1 to 50 ppm DDT," J. Pharmacol. Exper. Therap., 98:268 (1950).

195. Laws, E. R., Jr., A. Curley, and F. J. Biros, "Men With Intensive Occupational Exposure to DDT," Arch. Environ. Health, 15:766-65 (1967).

196. Lazutka, F. A., A. Vasilauskene, and Sh. G. Gefen, "Toxicological Evaluation of the Insecticide, Nicotine Sulfate," Gig. Sanit., 34:30-3; C.A. 71:21151f (1969).

197. Leeling, N. C., and J. E. Casida, J. Agr. Food Chem., 14:281; in O'Brien, Insecticides, p. 99 (1966).

198. Lehker, G. E., "Dictionary of Insecticides and Their Uses," Modern Sanitation and Building Maintenance, 10(3):13 (1958).

199. Lehman, A. J., "A Toxicological Evaluation of Household Insecticides," Assn. Food Drug Offic., U.S., Quarterly Bull., 18:3 (1954).

200. Lejhancova, M., "Damage to Skin by Mineral Oils," Prac. Lek., 20(4):164-8; C.A. 69:29928u (1968).

201. Lichtenstein, E. P., "Persistence and Behavior of Pesticidal Residues in Soils. Their Translocation Into Crops," Arch. Environ. Health, 10:825-6 (1965).

202. Lichtenstein, E. P., K. R. Schulz, R. F. Skrentny, and Y. Tsukano, "Toxicity and Fate of Insecticide Residues in Water," Arch. Environ. Health, 12:199-212 (1966).

203. Liebenow, W., E. Weber, and B. Schmidt, "Attempted Suicide With Metiram, a Fungicide Similar to Antabuse," Muenchen. Med. Wochenschr., 110:1238-41; C.A. 69:26250c (1968).

204. Lindahl, P. E., and K. E. Oberg, Exptl. Cell. Res., 23:228; in O'Brien Insecticides, p. 162 (1961).

205. Loiselle, D. W., "The Use of Chemical Weed Killers on Public Water Supply Watersheds," Jour. New Engl. Water Wks. Ass. 67:140 (1953).

206. Lutz-Ostertag, Y., R. Meiniel, and H. Lutz, "Effect of Parathion on the Development of Quail Embryos," Compt. Rend. Acad. Sci., Paris Ser. D, 268:2911-13; C.A. 71:48625a (1969).

207. Mackenthun, K. M., W. M. Ingram, and R. Porges, "Limnological Aspects of Recreational Lakes," Publ. Health Service Publ. No. 1167, 176 pp (1964).

208. Maier-Bode, H., "DDT in Body Fat of Humans," Med. Exp., 1:146 (1960).

209. Main, A. R., "The Role of A-Esterase in the Acute Toxicity of Paraoxon, TEPP, and Parathion," Can. J. Biochem. Physiol. 34:197-216 (1956).

210. Mamyan, D. B., "Primary Toxicological Characteristics of the Herbicide Diuron," Zh. Eksp. Klin. Med., 8(4):46-50; C.A. 70:114073b (1968).

211. Manigold, D. B., and J. A. Schulze, "Pesticides in Water," Pest. Monit. J., 3:124-135 (1969).

212. Mattson, A. M., and V. A. Sedlak, "Measurement of Insecticide Exposure," J. Agric. Food Chem., 8:107-110 (1960).

213. Matsumura, F., and C. T. Ward, "Degradation of Insecticides by the Human and Rat Liver," Arch. Environ. Health, 13:257-61 (1966).

214. McKee, J. E., "Report on Oily Substances and Their Effects on the Beneficial Uses of Water," Pub. 16, State Water Pollution Control Board, Sacramento, California (1956).

215. McKee, J. E., and H. W. Wolf, "Water Quality Criteria," Calif. State Water Qual. Control Board Publ. No. 3-A (1963).

216. McKennis, H., L. B. Turnbull, and E. R. Bowman, J. Amer. Chem. Soc., 81:3951; in O'Brien, Insecticides, p. 155 (1959).

217. Menusan, H., J. Econ. Entom., 41:302; in O'Brien, Insecticides, p. 149 (1948).

218. Mezentzeva, N. V., "Zinc Diisopropyl Dithiocarbamate," Toksikol. Nov. Khim. Veshchestv., Vnedryaemkh. Rezin. Shinnuyu Prom., 127-31:C.A. 71:48676t (1968).

219. Middleton, F. M., and J. J. Lichtenberg, "Measurements of Organic Contaminants in the Nation's Rivers," Ind. Eng. Chem. 52:99A-102A (1960).

220. Mikhailov, N. E., "Experimental Basis for Permissible Concentrations of Monuron and Diuron in Reservoir Waters," Prom. Zagryazneniya Vodoemov (8):72-4; C.A. 69:18215h (1967).

221. Milby, T. H., and W. L. Epstein, "Allergic Contact Sensitivity to Malathion," Arch. Environ. Health, 9:434-7 (1964).

222. Milby, T. H., F. Ottoboni, and H. W. Mitchell, "Parathion Residue Poisoning Among Orchard Workers," J. Am. Med. Assoc., 189:351-356 (1964).

223. Miller, C. W., W. E. Tomlinson, and R. L. Norgren, "Persistence of Parathion in Irrigation Waters," Pest. Monit. J., 1:47-48 (1967).

224. Mitchell, L. E., "Pesticides: Properties and Prognosis. Organic Pesticides in the Environment," Advances in Chemistry Series, Am. Chem. Soc., 60:1-22 (1966).

225. Mkhitarian, A. M., Izv. an. arm. SSR ser. tekn. nauk. 16 No. 4. 23-21. Monthly Index Russ. Acc. 16 1964. No. 10. 3108 (1963).

226. Moeller, H. C., and J. A. Rider, "Studies on the Anticholinesterase Effect of Parathion and Methyl Parathion in Humans," Fed. Proc., 20:434 (1961).

227. Moeller, H. C., and J. A. Rider, "Threshold of Incipient Toxicity of Systax and Methyl Parathion," Fed. Proc., 22:189 (1962).

228. Moore, E. W., Water and its Relation to Disease, Chap. 28, pp. 809-910.

229. Morgan, D. P., and C. C. Roan, "Renal Function in Persons Occupationally Exposed to Pesticides," Arch. Environ. Health 19:633-6 (1969).

230. Murphy, S. D., and K. L. Cheever, "Effect of Feeding Insecticides," Arch. Environ. Health, 17:749-58; C.A. 70:10583b (1968).

231. Nabb, D. P., W. J. Stein, and W. J. Hayes, "Rate of Skin Absorption of Parathion and Paraoxon," Arch. Environ. Health, 12:501 (1966).

232. Nakatsugawa, T., N. M. Tolman, and P. A. Dahm, "Degradation of Parathion in the Rat," Biochem. Pharmacol., 18:1103-14; C.A. 71:21147j (1969).

233. Narahashi, T., J. Cellular Comp. Physiol., 59:61; in O'Brien, Insecticides, p. 168-9 (1962).

234. Neal, P. A., T. R. Sweeney, S. S. Spicer, and von Oettinger, "The Excretion of DDT 2,2-bis-(p-chlorophenyl)-1,1,1, trichloroethane in Man Together with Clinical Observations," Public Health Repts., 61:403-409 (1946).

235. Gochfield, M., "Marine Contamination by Pesticides," New England J. of Med., 280:899-900 (1969).

236. Nicholson, H. P., "Water Contamination by Pesticides," J. Southeast Sect., Amer. Water Wks. Assoc., 32:8-20; C.A. 69:109637p (1968).

237. O'Brien, R. D., Insecticides. Action and Metabolism, Academic Press, New York, p. 108 (1967).

238. O'Brien, R. D., Insecticides. Action and Metabolism, Academic Press, New York, p. 291-306 (1967).

239. Olefir, A. I., and V. Kh. Vinogradova, "Embryotropic Effect of the Pesticides Sevin and Cyram," Vrach. Delo (11):103-6; C.A. 70:19205n (1968).

240. Ortelee, M. F., "Study of Men with Prolonged Intensive Occupational Exposure to DDT," AMA Arch. Industr. Health, 18:433-40 (1958).

241. Ottoboni, A., "Effect of DDT on Reproduction in the Rat," Toxicol. Appl. Pharmacol., 14:78-81; C.A. 70:86553n (1969).

242. Pallade, S., C. Popovici, G. Rotaru, E. Gabrielescu, and M. Dorobantu, "Action of Aldrin on Liver," Med. Lav., 59:346-56; C.A. 70:19192f (1968).

243. Pelikan, Z., K. Halacka, M. Polster, and E. Cerny, "Long-Term Poisoning in the Rat From Small Doese of Heptachlor," Arch. Belg. Med. Soc. Hyg. Med. Leg., 26:529-38; C.A. 71:2474x (1968).

244. Perron, R. C., and B. F. Barrentine, "Human Serum DDT Concentration Related to DDT Exposure," Arch. Environ. Health, 20:368-376 (1970).

245. Perry, A. S., A. M. Mattson, and A. J. Buckner, J. Econ. Entomol., 51:346; in O'Brien, Insecticides, p. 140 (1958).

246. Perry, A. S., S. Miller, and A. J. Buckner, J. Agr. Food Chem., 11:457; in O'Brien, Insecticides, p. 129 (1963).

247. Pestova, A. G., "Toxicity of Diptal and Avadex (Diallate)," Gig. Toksikol. Pestits. Lkin. Otravlenii (4):166-9; C.A. 70:2642e (1966).

248. Peterson, J. E., and W. H. Robinson, Toxicol. Appl. Pharmacol. 6:321; in O'Brien, Insecticides, p. 126 (1964).

249. Petty, C. S., A. Hedmeg, W. H. Reinhart, E. J. Moore, and L. P. Dunn, "Organic Phosphate Insecticides - A Survey of Blood Cholinesterase Activity of Exposed Agricultural Workers in Louisiana 1957," Am. J. Publ. Health, 42:62-9 (1959).

250. Pillsbury, D. M., W. B. Shelley, and A. M. Kligman, Dermatology, W. B. Saunders Co., Philadelphia, 1277 (1957).

251. Pinto, S. S., and B. M. Bennett, "Effect of Arsenic Trioxide Exposure on Mortality," Arch. Environ. Health, 7:583-91 (1963).

252. Polishuk, Z., M. Wassermann, D. Wassermann, Y. Groner, S. Lazarovici, and L. Tomatis, "Effect of Pregnancy on Storage of Organochlorine Insecticides," Arch. Environ. Health, 20:215-217 (1970).

253. Popadopoulos, N. M., and J. A. Kiatzios, J. Pharmacol. Exptl. Therap., 140:269; in O'Brien, Insecticides, p. 155-6 (1963).

254. Princi, F., and G. H. Spurbeck, "A Study of Workers Exposed to the Insecticides Chlordan, Aldrin, Dieldrin," Arch. Ind. Hyg. Occup. Med., 3:64-72 (1961).

255. Przezdziecki, Z., J. Bankowska, W. Malewska, and T. Janicka, "Subchronic Toxicity of Zineb, Maneb. and Captan," Rocz. Panstw. Zakl. Hig., 20:133-40 (1969).

256. Proklina-Kaminskaya, T. L., "Effect of Sevin and TMTD on the Activity of Mitochondrial Oxidative Enzymes," Farmakol. Toksikol. (Kiev) 144-6; C.A. 71:69654z (1968).

257. Proklina-Kaminskaya, T. L., "Effect of Carbamates on Tissue Respiration and the Activity of Cytochrome Oxidase and Succinate Dehydrogenase in Rat Liver," Farmakol. Toksikol. (Kiev) 153-5; C.A. 71:69655a (1968).

258. Quinby, G. E., J. F. Armstrong, and W. F. Durham, "DDT in Human Milk," Nature 207:726-28 (1965).

259. Quinby, G., and G. B. Clappison, "Parathion Poisoning," Arch. Environ. Health 3:538-42 (1961).

260. Quinby, G. E., W. J. Hayes, Jr.; J. F. Armstrong, and W. F. Durham, "DDT Storage in the U.S. Population," J. Amer. Med. Assoc., 191:109-13 (1965).

261. Quinby, G. E., and A. B. Lemmon, "Parathion Residues as a Cause of Poisoning in Crop Workers," J.A.M.A. 166:740-748 (1958).

262. Quinby, G. E., W. J. Hayes, J. F. Armstrong, and W. F. Durham, "DDT Storage in the U.S. Population," J. Amer. Med. Assoc., 191:175-9 (1965).

263. Quinby, G. E., and G. M. Doornink, "Tetraethyl Phrophosphate Poisoning Following Airplace Dusting," J. Amer. Med. Assoc., 191:95-100.

264. Read, S., and W. P. McKinley, "DDT and DDE Content of Human Fat," Arch. Environ. Health, 3:209-11 (1961).

265. Rider, J. A., H. C. Moeller, E. J. Puletti, and J. I. Swader, "Toxicity of Parathion, Systox, Octamethylphosphoramide, and Methyl Parathion," Toxicol. Appl. Pharmacol., 14:603-11; C.A. 71:2517p (1969).

266. Robeck, G. G., K. A. Dostal, J. M. Cohen, and J. F. Kreisel, "Effectiveness of Water Treatment Processes in Pesticide Removal," J. Amer. Water Wks. Assn., 57:1729-32 (1959).

267. Robinson, J., and C. G. Hunter, "Organochlorine Insecticide Content of Human Adispose Tissue in Southeastern England," Brit. J. Industr. Med. 22:220-9 (1965).

268. Robinson, J., and C. G. Hunter, "Organochlorine Insecticides: Concentration in Human Blood and Adipose Tissue," Arch. Environ. Health, 13:558-63 (1966).

269. Rosen, A. A., and F. M. Middleton, "Chlorinated Insecticides in Surface Waters," Anl. Chem., 31:1729-32 (1959).

270. Rudd, R. L., and R. E. Genelly, "Pesticides: Their Use and Toxicity in Relation to Wildlife," Calif. Dept. of Fish and Game, Game Bull. No. 7; in McKee, J. E., Water Quality Criteria, p. 362 (1956).

271. Ryazanova, R. A., "Health Evaluation of Agricultural Products Treated With Ziram and Zineb," Vop. Gig. Pitan. 32-8; C.A. 71:2469z (1967).

272. Ryzhenko, S. M., "Toxicity of Tselatox (Zelatox)," Gig. Toksikol. Pestits. Klin. Otravlenii (4):257-9; C.A. 69:42942p (1966).

273. Sartwell, P. E., Preventative Medicine in Public Health, 9th ed. Appleton, Century, Craft., 809-810 (1965).

274. Sax, N. I., Dangerous Properties of Industrial Materials, Reinhold Publishing Corporation, New York (1963).

275. Seifert, P., "Giftmord mit E-605 an Einem Sangling," Arch. Toxikol., 15:80 (1954).

276. Selby, L. A., K. W. Newell, C. Waggenspack, G. A. Hauser, and G. Junker, "Estimating Pesticide Exposure in Man as Related to Measurable Intake," Amer. J. Epidem., 89:241-253 (1969).

277. Shibley, G. J., "Volatile Liquid Petroleum Distillates," J. Amer. Med. Assoc., 186:93 (1963).

278. Shugaev, B. B., "Concentrations of Hydrocarbons in Tissues as a Measure of Toxicity," Arch. Environ. Health, 18:878-82; C.A. 71:47776p (1969).

279. Simmons, S. W., "Living Labs That Study How Pesticides Affect Man," FDA Papers (5) 13-16 (1969).

280. Smith, O. M., "The Detection of Poisons in Public Water Supplies," Water Wks. Eng., 97:1293; Water Poll. Abs. 18, Jan. 1945 (1945).

281. Smith, R. B., Jr., "Safe Use of Pesticides in Food Production," Arch. Environ. Health, 9:634-8 (1964).

282. Springer, P. F., North Carolina Pesticide Manual, North Carolina State College, Raleigh, p. 87; in McKee, Water Quality Criteria, p. 367 (1957).

283. Stammers, F. M. G., and F. G. S. Whitfield, "The Toxicity of DDT to Man and Animals," Bul. Entomol. Rsch., 38:1-73 (1947).

284. Stavinoha, W. B., J. A. Rieger, Jr., L. C. Ryan, and P. W. Smith, "Effects of Chronic Poisoning by an Organophosphorus Cholinesterase Inhibitor on Acetylcholine and Norepinephrine Content of the Brain," Organic Pesticides in the Environment, Advances in Chemistry Series, Amer. Chem. Soc., 60:79-88 (1966).

285. Stecher, P. G., ed., The Merck Index, 8th ed., Merck and Co., Rahway, N. J. (1968).

286. Stein, A. A., D. M. Serrone, and F. Coulston, "Safety Evaluation of Methoxychlor in Human Volunteers," Toxic. Appl. Pharmacol., 7:499 (1965).

287. Sternburg, J., and C. W. Kearns, "Presence of Toxins Other Than DDT in the Blood of DDT-Poisoned Roaches," Science 116:144 (1952).

288. Stevenson, D. E., and A. I. T. Walker, "Hepatic Lesions Produced in Mice by Dieldrin and Other Hepatic Enzyme-Inducing Compounds," J. Eur. Toxicol., 2:83-4; C.A. 71:6896p (1969).

289. Stoner, H. B., Nature, 172:1044; in O/Brien, Insecticides, p. 297 (1953).

290. Tanimura, T., T. Katsuya, and H. Nishimura, "Embryotoxicity of Acute Exposure to Methyl Parathion in Rats and Mice," Arch. Environ. Health, 15:609-13 (1967).

291. Tarrant, K. R., and J. O'G. Tatton, "Organochlorine Pesticides in Rainwater in the British Isles," Nature, 219:725-7 (1968).

292. Taylor, F. B., "Significance of Trace Elements in Public, Finished Water Supplies," J. Amer. Water Wks. Assoc., 55:619-23 (1963).

293. Terada et al, "Arsenic Poisoning From Water Pollution," J. Amer. Med. Assoc., 181:803 (1962).

294. Tinsley, I. J., "Interaction of Dieldrin with Thiamine," Proc. Soc. Exp. Biol. Med., 129:463-5; C.A. 70:27977 (1968).

295. Tischler, N., J. Econ. Entom., 28:215; in O'Brien, Insecticides, p. 161 (1936).

296. Treon, J. F., and F. P. Cleveland, J. Agr. Food Chem., 3:402 (1955).

297. Tzukamoto, M., Botyu-Kagaku 24:141; in O'Brien, Insecticides, p. 129 (1959).

298. Van der Weij, H. G., "Developments and Investigations in the Chemical Control of Aquatic Weeds in the Netherlands," Proc. Brit. Weed Contr. Conf., 8th 1966, 3:835-41; C.A. 71:29405n (1966).

299. Van Horn, W. M., "Possible Stream Pollutional Aspects of Mill Antiseptics," Paper Trade Jour. 117:24, 33; in McKee, Water Quality Criteria, p. 217 (1943).

300. Van Horn, W. M., and R. Balch, TAPPI 38:151; C.A. 49:7853; in McKee, Water Quality Criteria, p. 217 (1955).

301. Villeneuve, D. C., G. Mulkins, K. A. McCully and W. P. McKinley, "Inhibition of Beef Liver Hydrolytic Enzymes by Organophosphorus Pesticides. Comparison of the Effect of Several Pesticides and Their Oxons on the Inhibition Response," Bull. Environ. Contam. Toxicol., 439-47; C. A. 70:85805j (1969).

302. von Frank, A. J., "Where Are Water Quality Standards Heading?" Water and Wastes Eng., 6(5):16-18.

303. Vrochinskii, K. K., S. S. Grebenyuk, and A. L. Burshtein, "Contamination of Water Reservoirs With Pesticides," Gig. Sanit., 33(11):69-72; C.A. 70:22783e (1968).

304. Wade, L., "Observations on Skin Cancer Among Refinery Workers," Arch. Environ. Health, 6:730-5 (1963).

305. Warren, M. C., J. P. Conrad, J. J. Bocian, and M. Hayes, "Clothing-Borne Epidemic," J. Amer. Med. Assoc., 184:266-8 (1963).

306. Wasserman, M., M. Gon, D. Wasserman, and L. Zellermayer, DDT and DDE in the Body Fat of People in Israel," Arch. Environ. Health, 11:375-9 (1965).

307. Weibel, S. R., R. B. Weidner, J. M. Cohen, and A. G. Christianson, "Pesticides and Other Contaminants in Rainfall and Runoff," J. Amer. Water Wks. Assoc., 58:1074-84 (1966).

308. Weir, P. A., and C. H. Hine, "Effects of Various Metals on Behavior of Conditioned Goldfish," Arch. Environ. Health, 20:45-51 (1970).

309. Welch, R. M., and J. M. Coon, "Studies on the Effect of Chlorcyclizine and Other Drugs on the Toxicity of Several Organophosphate Anticholinesterases," J. Pharmacol. Exptl. Therap., 143:192 (1964).

310. West, I., "Pesticides as Contaminants," Arch. Environ. Health, 9:626-33 (1964).

311. West, I., "Biological Effects of Pesticides in the Environment, Organic Pesticides in the Environment," Advances in Chemistry Series, Amer. Chem. Soc., 60:47-53 (1966).

312. Wheatley, G. A., and J. A. Hardman, "Indications of the Presence of Organochlorine Insecticides in Rainwater in Central England," Nature, 207:486-7 (1965).

313. Whitmore, F. C., Organic Chemistry, 2nd Ed., Van Nostrand, New York (1951).

314. Wigglesworth, V. B., "A Case of DDT Poisoning in Man," Brit. Med. J. p. 517 (April 14, 1945).

315. Wolfe, H. R., et al., "Health Hazards of Discarded Pesticide Containers," Arch. Environ. Health, 3:45-51 (1961).

316. Zamfir, Gh., V. Nastase, M. Srainer, S. Freund, L. Alexa, S. Apostol, and N. Topala, Igiena 17:405-13; C.A. 71:21152g (1968).

317. Zavon, M. R., "Danger of Pesticides to the Liver," J. Amer. Med. Assoc., 191:419 (1965).

318. Zavon, M. R., C. H. Hine, and K. D. Parker, "Chlorinated Hydrocarbon Insecticides in the Human Body Fat in the United States," J. Amer. Med. Assoc., 193:837-9 (1966).

C. BIBLIOGRAPHY OF STATE RECREATIONAL WATER QUALITY DOCUMENTS

BIBLIOGRAPHY

1. Anon. Alaska State Plan Water Quality Standards. Dept of Health and Welfare, 6-40 (1967).

2. Anon. Water Quality Standards for Surface Waters in Arizona, State Dept. of Health, 1-55 (1968).

3. Anon. Water Quality Criteria. Water Improvement Commission State of Alabama, 9-11 (1967).

4. Anon. Outdoor Bathing Places. Arkansas State Board of Health, 4 (1964).

5. Anon. Regulation No. 2. State of Arkansas Pollution Control Commission, 1-7 (1967).

6. Anon. Laws and Regulations Relating to Ocean Water-Contact Sports Areas. State of California Dept. of Public Health, 176 (1958).

7. Anon. Water Quality Standards for Colorado. Colorado Dept. of Health, 4 (1968).

8. Anon. . Water Quality Criteria. Connecticut State Dept. of Health, 2-4.

9. Anon. Water Quality Standards. Delaware Water and Air Resources Commission State of Delaware, 1-33 (1968).

10. Anon. Basin Regulations - Water Quality. Delaware River Basin Commission, 1-8 (1968).

11. Anon. Revised Water Quality Criteria and Uses. District of Columbia, 1-4 (1968).

12. Anon. Rules of the State Board of Health. Florida Sanitary Code, 21-36.

13. Anon. Standards of Water Quality for Water of the Territory of Guam. Water Pollution Control Commission. Territory of Guam, 7-8 (1968).

14. Anon. Public Health Regulations. Dept. of Health, State of Hawaii, Chap. 37-A, 1-8 (1968).

15. Anon. Rules and Regulations for the Establishment of Standards of Water Quality. Idaho State Board of Health, 2-5 (1968).

16. Anon. Rules and Regulations for Standards of Water Quality for the Interstate Waters of Idaho. Idaho State Board of Health, 1-9 (1967).

17. Anon. Implementation, Enforcement and Surveillance Plan. Idaho Department of Health (1967).

18. Anon. Rules and Regulations SWB-7-15. Water Quality Standards. Illinois Sanitary Water Board (1968).

19. Anon. Regulations SPC4-9. Stream Pollution Control Board, State of Indiana (1967).

20. Anon. Regulations SPCIR, Water Quality Standards for Waters of Indiana. Indiana Stream Pollution Control Board, 1-4 (1967).

21. Anon. Tri-State Compact for Pollution Abatement. Interstate Sanitation Commission, 4-5 (1941).

22. Anon. River Basin Water Quality Criteria. Kansas State Board of Health, 1-76 (1968).

23. Anon. Water Quality Standards for Interstate Waters. Water Pollution Control Commission. (1969).

24. Anon. Water Quality Criteria and Plan for Implementation. State of Louisiana, Louisiana Stream Control Commission, 7-85. (1968).

25. Anon. Rules and Regulations of the Department of Health and Welfare Relating to Swimming Pools and Bathing Beaches. Dept. of Health and Welfare, 1-7. (1956).

26. Anon. Water and Air Environmental Improvement Commission, Revised Statutes of 1964. 3-48. (1967).

27. Anon. Water Resources Regulation 4.8. State of Maryland Water Resources Commission and Dept. of Water Resources, 1-11. (1969).

28. Anon. Water Quality Standards. Commonwealth of Massachusetts Water Resources Commission. Division of Water Pollution Control, 1-4. (1967).

29. Anon. Summary of Comments on the Corrections to the Proposed Water Quality Criteria and Plans for Implementation for Michigan Waters. State of Michigan Water Resources Commission, 25-41. (1967).

30. Anon. Water Quality Standards for Michigan Waters. State of Michigan Water Resources Commission, Dept. of Conservation, 6-21. (1967).

31. Anon. Water Quality Standards for Michigan Intrastate Waters. Michigan Water Resources Commission, Dept. of Conservation, 18-21. (1968).

32. Anon. State of Mississippi Water Quality Criteria for Interstate and Coastal Waters. Mississippi Air & Water Pollution Control Commission, 1-5.(1968).

33. Anon. State of Mississippi Air and Water Pollution Control Act. Mississippi Air and Water Pollution Control Commission. 8-9. (1966).

34. Anon. A Guide for the Design and Operation of Public Bathing Places. Dept. of Public Health and Welfare of Missouri, Division of Health, 11. (1967).

35. Anon. Lower Mississippi River Basin, Southwest Lower Mississippi Water Quality Standards. Missouri Water Pollution Board. (1968).

36. Anon. Upper Mississippi River Basin, Southwest Lower Mississippi Water Quality Standards. Missouri Water Pollution Board. (1968).

37. Anon. Upper Mississippi Water Quality Standards. Missouri Water Pollution Board. (1968).

38. Anon. Salt River and Tributaries, Mississippi River Tributaries and Des Moines River, Water Quality River. Missouri Water Pollution Board. (1968).

39. Anon. White River Basin Southwest - Lower Mississippi Water Quality Standards. Missouri Water Pollution Board. (1968).

40. Anon. Grand (Neosho) River Basin Southwest - Lower Mississippi Water Quality Standards. Missouri Water Pollution Board. (1968).

41. Anon. Missouri River Basin, Water Quality Standards. Missouri Water Pollution Board. (1968).

42. Anon. Osage-Gasconade River Basin, Missouri River Basin Water Quality Standards. Missouri Water Pollution Board. (1968).

43. Anon. Grand-Chariton River Basin, Missouri River Basin Water Quality Standards. Missouri Water Pollution Board. (1968).

44. Anon. Minor Missouri River Tributaries Water Quality Standards. Missouri Water Pollution Board. (1968).

45. Anon. Big Blue River Water Quality Standards. Missouri Water Pollution Board. (1968).

46. Anon. Water Quality Criteira. State of Montana Water Pollution Control Council, 1-11. (1967).

47. Anon. Water Pollution Control Act. State of Nebraska Chap. 71, Article 30, Statutes of Nebraska. (1967).

48. Anon. Water Quality Standards. Water Pollution Control Council, 3-11. (1969).

49. Anon. Water Pollution Control Regulations. Bureau of Environmental Health, 13-61. (1967).

50. Anon. State of New Hampshire, Laws Relating to the Water Supply and Pollution Control Commission. Water Supply and Pollution Control Commission.

51. Anon. Rules and Regulations Establishing Classifications to be Assigned to the Waters of This State and Standards of Quality to be Maintained in Waters so Classified. New Jersey State Dept. of Health. (1967).

52. Anon. New Mexico Water Quality Control Commission Ammendments to the Water Quality Standards. Water Quality Control Commission, 1-5. (1968).

53. Anon. Water Quality Standards. New Mexico Water Quality Control Commission. (1967).

54. Anon. Classifications and Standards of Quality and Purity for Waters of New York State. New York State Dept. of Health, 506-507. (1968).

55. Anon. Classifications and Water Quality Standards Applicable to the Surface Waters of North Carolina. Dept. of Water and Air Resources, 10-11.

56. Anon. Water Quality Standards for Surface Waters of North Dakota. North Dakota State Dept. of Health. (1967).

57. Anon. ORSANCO Nineteenth Yearbook. Ohio River Valley Water Sanitation Commission, 12-29. (1967).

58. Anon. Bacterial-Quality Objectives for the Ohio Rover. Ohio River Valley Sanitation Commission, 21-26. (1951).

59. Anon. Oklahoma's Water Quality Standards. Oklahoma Water Resources Board. (1968).

60. Anon. Oklahoma Public Bathing Place Interpretive Code. Environmental Health Services, State Dept. of Health. (1965).

61. Anon. Standards for Public Water Supply Facilities. Environmental Health Services, State Dept. of Health, Oklahoma. (1964).

62. Anon. Rules of the Georgia Water Quality Control Board. Water Quality Control Board, 15-23. (1965).

63. Anon. Georgia Water Quality Control Act. Water Quality Control Board. (1966).

64. Anon. Fourth Annual Progress Report - 1968. Georgia Water Quality Control Board. (1968).

65. Anon. Iowa Water Pollution Control Law. Iowa Water Pollution Control Commission. (1966).

66. Anon. Water Pollution Control Progress Report. Iowa Water Pollution Control Commission. (1968).

67. Anon. Water Quality Criteria and Plan for Implementation and Enforcement for the Surface Waters of Iowa. Water Pollution Control Commission, 9-15. (1967).

68. Anon. Oregon Administrative Rules, CH 334. State Sanitary Authority, 1-29. (1967).

69. Anon. Act No. 142, Water Pollution Control Act. Dept. of Health, Puerto Rico. (1950).

70. Anon. Sanitary Regulations No. 127, 128, 129, 131. Dept. of Health, Puerto Rico. (1967).

71. Anon. Water Classification - Standards System for the State of South Caroline. South Carolina Pollution Control Authority, 1-8. (1967).

72. Anon. Classes and Standards for Tidal Salt Waters. South Caroline Pollution Control Authority, 1-3. (1967).

73. Anon. Water Quality Standards for the Surface Waters of South Dakota. South Dakota Committee on Water Pollution, 1-28. (1967).

74. Anon. General Water Quality Criteria for the Definition and Control of Pollution in the Waters of Tennessee. Tennessee Stream Pollution Control Board, 4-5. (1967).

75. Anon. Regulations Governing the Construction, Operation and Maintenance of Organized Camps in Tennessee. Tennessee Dept. of Public Health, 6-7. (1966).

76. Anon. The State of Texas Water Quality Requirements, Vol. 1, Inland Waters. Texas Water Quality Board. (1967).

77. Anon. The State of Texas Water Quality Requirements, Vol. 2, Coastal Waters. Texas Water Quality Board. (1967).

78. Anon. Code of Waste Disposal Regulations - Part II - Standards of Quality for Waters of the State. Utah Water Pollution Control Board, 1-12. (1968).

79. Anon. Chap. 7 - Water Pollution Control. Water Quality Standards for Coastal Waters of the Virgin Islands. Office of the Commissioner of Health. (1968).

80. Anon. Water Use Classes, Standards of Quality and Technical Guidelines for Intrastate Waters. State of Vermont Dept. of Water Resources. (1967).

81. Anon. General Policy, Water Use Classes and Standards of Quality for Intrastate Waters. State of Vermont Dept. of Water Resources. (1967).

82. Anon. A Regulation Relating to Water Quality Standards for Interstate and Coastal Waters of the State of Washington. Water Pollution Control Commission, 1-7. (1967).

83. Anon. Information Bulletin Public Hearings on Proposed Water Quality Standards for Intrastate Waters. Washington State Water Pollution Control Commission. State of Washington. (1969).

84. Anon. A Proposed Regulation Relating to Water Quality Standards for Interstate Waters. Water Pollution Control Commission, State of Washington, 3-8. (1969).

85. Anon. State of West Virginia Administrative Regulations, Water Quality Criteria on Inter- and Intrastate Streams. Division of Water Resources, 12-55. (1965).

86. Anon. Implementation Plan for West Virginia Water Quality Standards. Division of Water Resources. (p968).

87. Anon. Wisconsin Water Quality Standards. Resource Development Board, 1-3. (1968).

88. Anon. Minnesota Administrative Rules and Regulations, WPC 1-21. Minnesota Pollution Control Agency. (1968).

89. Anon. Water Quality Standards for Interstate Waters in Wyoming. Wyoming Dept. of Health, 5-14. (1968).

90. Barker, Blaine B. Water Pollution Control Section. South Dakota Department of Health. Personal Communication. (1969).

91. Boardman, Richard M. Pennsylvania Department of Health. Division of Water Quality. Personal Communication. (1969).

92. Boes, Robert J. Ohio River Valley Water Sanitation Commission. Personal Communication. (1969).

93. Brinck, Claiborne W. Division of Environmental Sanitation. State of Montana. Personal Communication. (1969).

94. Filipi, T. A. Nebraska Water Pollution Control Council. Personal Communication. (1969).

95. Garner, L. M. Missouri Division of Health. Personal Communication.(1969).

96. Glenn, Thomas R. Jr. Interstate Sanitation Commission. Personal Communication. (1969).

97. Gregory, E. G. Bureau of Environmental Health. State of Nevada. Personal Communication. (1969).

98. Harmeson, Donald K. Bureau of Environmental Health. State of Delaware. Personal Communication. (1969).

99. Kinniburgh, Richard S. Water Supply and Pollution Control Commission. State of New Hampshire. Personal Communication. (1969).

100. Long, Emory G. Texas Water Quality Board. Personal Communication. (1969).

101. Martin, Walter C. Kentucky State Water Pollution Commission. Personal Communication. (1969).

102. Miller, Robert S. Bureau of Water Supply. Missouri Division of Health. Personal Communication. (1969).

103. Ongerth, H. J. Bureau of Sanitary Engineering. Personal Communication. (1969).

104. Peterson, Norman L. North Dakota Department of Health. Division of Water Supply and Pollution Control. Personal Communication. (1969).

105. Pine, Roland E. Washington State Water Pollution Control Commission. Personal Communication. (1969).

106. Plews, Gary. Recreation and Housing Section. Washington State Department of Health. Personal Communication.

107. Poole, B. A. Stream Control Board. State of Indiana. Personal Communication. (1969).

108. Quisenberry, Walter B. Department of Health, Hawaii. Personal Communication. (1969).

109. Rhame, George A. Pollution Control Authority. South Carolina State Board of Health. Personal Communication. (1969).

110. Rice, R. G. Department of Public Health. State of Michigan. Personal Communication. (1969).

111. Rozich, Frank J. Water Pollution Control Division. State of Colorado Department of Health. Personal Communication. (1969).

112. Schraufnagel, Francis H. Bureau of Standards and Water Surveys. State of Wisconsin Department of Natural Resources, Personal Communication. (1969).

113. Segessor, Ernest R. New Jersey State Department of Health. Personal Communication. (1969).

114. Shea, W. J. Rhode Island Department of Health. Personal Communication. (1969).

115. Speiser, Arnold. Water Quality Control Division. District of Columbia. Personal Communication. (1969).

116. Trygg, John E. Louisiana State Department of Health. Personal Communication. (1969).

117. Vasuki, N. C. State of Delaware Water and Air Resources Commission. Personal Communication. (1969).

118. von Frank, A. J. Where Are Water Quality Standards Heading? Water and Wastes Engineering/Industrial. 16-18. (1969).

119. Williamson, Arthur E. Sanitary Engineering Services. Wyoming State Department of Health. Personal Communication. (1969).

120. Woodhull, Richard S. Environmental Health Services Division. State of Connecticut. Personal Communication. (1969).

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